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TEXTBOOK OF DIETETICS

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CENTRAL FOOD TECHNOLOGICAL RESEARCH
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FOREWORD

IEDICINE can be regarded as the application of biological science or the prevention and cure of disease. In recent years, biological cience has been advancing so rapidly that it must be difficult for he general practitioner, and even for the clinical teacher, to keep breast with the growing volume of knowledge which is of use to he profession.

The difficulty is increased by the fact that knowledge acquired in iological research is seldom directly applicable to human beings. his is well illustrated in the case of nutrition. Our knowledge of he requirements for vitamins and minerals has come mainly from esearches on animals. We know with great exactitude the requirements of the rat for the main essential food constituents; we know he pathological conditions which will develop in deficiencies of hese, and in some cases we can predict the date at which they ill appear. But, in experiments with rats, the research worker eals with a standard animal of fixed heredity, fixed environmental onditions and known absence of previous nutritional or infective iseases which might complicate the result of the experiment. The esults of scientific research, obtained under these conditions, annot be applied directly to human beings who vary widely in heir hereditary make-up, in their environment and in their past ealth record. All of these affect their dietary requirements, which, any case, are not identical with those of the rat.

The position is further complicated by psychological factors. It common knowledge that fear and worry upset digestion. The etabolic processes are also affected by the emotions. Some of the onditions which these emotions produce are indistinguishable from e which arise from deficiencies in the diet. On the other hand, etary deficiencies aff t the central nervous system. Listlessness d depression which may occur, for example, in nutritional æmia and in vitamin B_1 deficiency are not distinguishable from onditions which may arise from fear and worry. In many cases f indefinite ill-health, it is often impossible to assess the relative mportance of nutritional and psychological causes.

It is obvious that there must be a time-lag between the acquisiion of fundamental scientific knowledge in the laboratory and it on of pplication in human medicine, where so many ot r factors nfusion, e taken into account. The time-lag has in the been his is du the fact that most of th nutrition were either biochemists without a medical training c medical men who devoted themselves so exclusively to scientif research that they lost touch with general clinical medicine. Ther is need for closer co-operation between the research worker and th clinician. This co-operation is now taking place to an increasin extent in many medical schools with highly successful results.

Even after clinical research has adapted the new knowledge t the needs of medicine, there is a further time-lag in getting i applied by the general practitioner, who cannot find time to kee in touch with the recent advances in nutritional science. Ther are, of course, many books on nutrition and dietetics, but they ar mostly based on the results of laboratory research. What the bus practitioner needs is a book which gives in concise form th established facts of practical importance in the prevention an treatment of disease by diet. This book meets these needs. It deal lucidly with the new knowledge of nutrition and its application i medicine, and it is written by physicians who have themselves don original research on nutrition in relation to health and disease. have pleasant recollections of the work on nutritional anæmias vitamin deficiencies in children, and the role of nutrition i rheumatism, which was carried out jointly by the authors and m colleagues and myself at the Rowett Institute.

It is to be hoped that the physician will not use this book merel as a textbook of references in the treatment of disease. The promotion of health is now rightly regarded as being of as muc importance as the cure of disease. There is no longer any doub that the diet of a large part of the population is not fully adequat for health. The medical practitioner must play the leading par in the improvement of national health by proper feeding. He i in direct touch with the homes of the people, and he understand far better than the laboratory worker or government offici economic and environmental conditions and the psychologica factors which must be taken into account when giving advice o diet which will be effective in improving the health of the family

This book can be strongly recommended to the practitioner. He will find in it not only precise and detailed guidance for dieteti treatment, but, what is equally important, a scientific and lucid account of the recently acquired knowledge on nutrition on which uch treatment, whether for the promotion of health or the ntion and control of established disease, must be based.

JOHN BOYD ORR

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PREFACE TO THE SECOND EDITION

The six years that have elapsed since the first publication of this book have seen great developments both in the fundamentals of nutrition and in their application to everyday dietetic and clinical problems. Unfortunately, the assimilation of these advances into the substance of the text has involved a not inconsiderable increase in its length; nevertheless, the original objectives and structure (as described in the preface to the first edition) have been left intact. The work is still directed primarily to students and general practitioners, for we hold most strongly that the family doctor, properly instructed, can and should play the chief part in the education of the public on matters of diet and nutrition. And though there is more to impart than ever before, it can be said truly that further knowledge has led to simplification rather than to complication of the subject. As the physiological foundations become firmer the student will find that the erection of the clinical superstructure becomes an easier, more profitable and more absorbing occupation.

A few words must be said about the differences between the two editions:

(1) The section on the Physiology of Nutrition takes full account of the many advances in knowledge made since the publication of the first edition in 1940. The most fundamental revisions are in the chapter on Proteins (which now includes a full account of protein hydrolysates) and in the necessarily much enlarged chapter on Vitamins (which discusses, among other new material, the use of folic acid in the macrocytic anæmias).

(2) The chapter on Nutrition and Diet in War-time has been completely re-written in the light of the experience of the past few years. It examines the Government's nutritional policy and the contribution this has made to the maintenance of the nation's health.

(3) There is a new section on the Treatment of Starvation. This subject came into prominence through the tragic events in concentration camps and in countries occupied by Germany and Japan, but there are ominous signs that the problem may remain with us, at least in some regions of the world, for some years to come.

(4) A new chapter has been included on Special Feeding Methods, parenteral and enteral, the former involving the introduction of nutrients by subcutaneous, intramuscular or intravenous infusion,

and the latter by gavage, duodenal and jejunal feeding, gastrostomy feeding, or by the nutrient enema. All these methods are described in sufficient detail to enable the practitioner to supply fluid and nourishment to patients who cannot take them in normal ways.

(5) The section dealing with Tables, Recipes and Diet Sheets has been materially altered. The tables of individual nutrients previously dispersed through the book are now replaced by a comprehensive and greatly extended Table of the Nutritive value of Foodstuffs based on the most recent authoritative figures published in Great Britain and the United States. This table offers a detailed analysis of a great diversity of foods, raw and cooked, setting out their content of all the components whose quantitative requirements for human nutrition have been clearly established. Some common foods used in tropical countries are also included, for the nutritional problems of these regions are in general more pressing than those of the western world.

This section also includes a new chapter on methods for carrying out dietetic surveys and for using food tables in the analysis and construction of diets. This is the only chapter which may be regarded as of more concern to the specialist than to the family doctor. Nevertheless, it should serve for the guidance of any practitioner faced with the problem of devising diets whether for the individual or for larger groups.

(6) A successful feature of the first edition was the inclusion of lists of exchanges for the avoidance of dietetic monotony, and of large numbers of diet sheets suitable for patients on different economic levels. These features have been retained in the present edition, but with all the amendments required to bring the tables and diets into line with the principles of treatment described and with the amended figures for food values.

(7) Finally, the chapters on the Treatment of Established Diseases have been brought up to date, supplemented by new data, and in one case (the chapter on diseases of the liver and gallbladder) completely rewritten. We may mention in particular new sections on the use of globin insulin, on the protein intake in hepatitis, on the role of choline and methionine in hepatic function, and further material (which confirms views expressed in the first edition) on the protein intake in nephritis, cholesteral metabolism in relation to cholecystitis, and the dietetic treatment of peptic ulcer and its complications.

Once again we wish to acknowledge with gratitude the help we have received from numerous colleagues and friends. In particular we would thank Dr. C. P. Stewart for his helpful criticism and advice on the chapters on the physiology of nutrition. We are also indebted to Miss E. M. Mackenzie for the time and care she has devoted to correcting proofs, and to Miss M. P. Russell, M.A., Assistant Librarian in the Royal College of Physicians, Edinburgh, for the preparation of the index.

L. S. P. Davidson.
Ian A. Anderson.

PREFACE TO THE FIRST EDITION

This textbook is based on courses of lectures on the physiology of nutrition and the practice of dietetics which have been given by the authors for several years to medical students in the University of Aberdeen. During this period these subjects have grown enormously in scope and importance; and with this development there has been an unfortunate tendency for the practice of dietetics to pass from the sphere of the general practitioner to that of the specialist. In fact, as will be shown in these pages, common-sense advice on diet and the application of dietetic measures for the promotion of health and the treatment of disease are well within the competence of doctors properly instructed in the physiology of nutrition. The distinction between the two subjects is very important. In nutritional experiments food analyses are essential, for the science of nutrition is concerned with stating in quantitative terms both the composition of foods and their fate in and action on the body; but in the dietetic treatment of disease the practitioner must be satisfied with approximations, bearing in mind that the constituents of food vary with such factors as the seasons, the climate, the soil and the method of cooking. Fortunately, precise estimates of food values are seldom called for in practice; and even the weighing and measurement of foods can usually be dispensed with, except perhaps in the treatment of diabetes mellitus and obesity. What is important, however, is a knowledge of what foods may be permitted and what may not, with the exact reasons for the concessions and the prohibitions. No dietetic restriction should be imposed that cannot be justified on biochemical, physiological, or clinical grounds. To withhold much-desired foods on no better sanction than fashion or tradition not only discredits the practice of dietetics but may inflict misery and harm on the suffering patient. For these reasons it has been one of our primary aims to set out the scientific principles on which our dietetic recommendations are based.

There are very few conditions in which dietetic treatment can be satisfactorily considered without relation to associated therapeutic measures. In some conditions – *e.g.* diabetes mellitus – the various forms of treatment are so closely interdependent that to separate dietetic treatment from the rest would be both arbitrary and unprofitable. In every condition discussed in this work we have endeavoured, while laying special stress on dietetic measures, to consider the treatment as a whole, even though this has involved frequent, though necessarily brief, references to such measures as psychotherapy, physiotherapy and the administration of drugs. The importance of correcting unphysiological methods of living and eating are repeatedly stressed, for failure in these respects may invalidate any benefits from the dietetic regime prescribed.

The work falls naturally into five parts:

I. A general survey of the nation's diet, past and present, with particular reference to the diet at various income levels and to experimental, clinical and sociological data on the relation of diet to health and fitness.

II. A simple but for clinical purposes complete account of the physiology of nutrition. In case this section should be regarded as a mere academic interlude – of concern to the student with examinations to pass but not to the doctor with patients to treat – it is worth re-emphasizing that without a satisfactory knowledge of the basic principles of nutrition it is impossible to give sound guidance on the use of dietetic measures for the maintenance of health or to institute rational dietetic treatment of disease. Much of the material in this section (particularly on the vitamins) is based on very recent experimental and clinical studies.

III. A discussion on diet in health, with particular reference to modifications in the diet that should be made in periods of stress. This section deals with the problems of diet in childhood, in pregnancy and lactation, and in athletic training; and it also includes a chapter on nutrition and diet in war-time.

IV. A full discussion of the dietetic treatment of established disease.

V. Tables, Recipes and Diet Sheets. The tables of food values are, we believe, wholly adequate for the purposes of the general practitioner. Lists of exchanges are included, so that monotony can be avoided and available foods substituted for those of which, in war-time, there may be a temporary shortage, or cheaper but dietetically equivalent foods used in place of those beyond the patient's means. In view of economic factors and differences in social custom, the Diet Sheets are given in duplicate, one for poor and the other for well-to-do patients.

No one writing on dietetics to-day can fail to have been influenced by such authorities as McLester, Sherman, McCance, Drummond, Mottram and Lusk; and to these and many other workers who have contributed to the common store of knowledge on the physiology of nutrition we gratefully acknowledge our indebtedness. We wish also to acknowledge with profound gratitude the help we have received from many colleagues and friends. In particular we would thank Professor D. M. Dunlop, Dr. C. P. Stewart, Dr. J. G. M. Hamilton, and Dr. B. Nicol of the University of Edinburgh, and Miss Leitch of the Rowett Research Institute, Aberdeen, for their help in reading the original manuscript and for invaluable suggestions which have been embodied in the text. To Mr. A. L. Bacharach we owe a special debt for expert advice and suggestions of which full use was made in the preparation of the chapter on vitamins; and to Dr. R. P. Cook of the School of Biochemistry, Cambridge, for data on the cholesterol content of foods and for other help in connexion with the section on Cholecystitis and Cholelithiasis. We would also express our warmest thanks to Miss E. M. Thomson, Sister Dietician at the Royal Infirmary, Aberdeen, for the preparation of the Diet Sheets, to Sister Buchan and Miss Wood of the Dietetic Department, Edinburgh, for help in the scrutiny of tables and recipes, and to Miss A. Inglis, Secretary in the Department of Medicine, Edinburgh, for invaluable and most detailed work in checking the text at every point. We are deeply grateful to Sir John Boyd Orr both for his kindness in writing a Foreword and for his generous appreciation of this work. Finally, our thanks are due to Miss Russell, Assistant Librarian in the Royal College of Physicians, Edinburgh, for the preparation of the Index, and to Dr. M. Newfield of Messrs. Hamish Hamilton for his unfailing co-operation and assistance.

It is our hope that this book will not only help the general practitioner to give sound advice on the relation of diet to the maintenance of health but supply him with the knowledge and the confidence to undertake the dietetic treatment of established disease.

L. S. P. DAVIDSON.
IAN A. ANDERSON.

PART I

INTRODUCTION

CHAPTER I

GENERAL SURVEY OF THE PROBLEM

THE rapid advance in the science of nutrition made in recent years has shown that diet has a profound influence in determining the state of health. It has been proved that much of the ill health which afflicts human populations can be attributed directly to deficiencies in diet, and there is good evidence to suggest that an increased susceptibility to certain infectious diseases, such as tuberculosis, may be conditioned by a faulty diet. It is therefore essential that the practising members of the medical profession should be familiar with the physiological principles of nutrition, in order that they may advise their patients how to prevent dietary deficiency and thus raise the national standard of health. In addition, dietetic treatment is important in certain forms of established disease, and the practitioner must be able to prescribe the diets which are most suitable for the treatment of these diseases, and which, at the same time, fulfil the fundamental nutritional requirements.

The first essential – the prevention of nutritional deficiency by the provision of an adequate diet – is to-day not so much a scientific as a social and economic problem, since most scientists, basing their views on the results of experiments and clinical observations, are in reasonably close agreement as to the quantities of the various known essential nutrients necessary to maintain optimal health. Numerous sets of standards have been published, an example of which is that drawn up in 1943 by the Food and Nutrition Board of the United States National Research Council (see Table 1). It is an established but not sufficiently well-recognized fact, however, that the income of a not inconsiderable fraction of the population of this country is too small to provide a dietary adequate in all respects.

If one considers average figures only, there has been an undoubted improvement in the national dietary, even within the last thirty years. Tables 2 and 3 show the changes in the amounts of

TABLE 1

QUANTITIES OF NUTRIENTS REQUIRED FOR INDIVIDUALS PER DAY

(Modified from *Recommended Dietary Allowances*, Food and Nutrition Board, U.S. National Research Council, 1943)

	Calories	Protein, grams	Calcium, mg.	Iron, mg.	Vitamin A, I.U.	Thiamin (Vitamin B_1)		Ribo-flavin, mg.	Niacin (Nicotinic acid) mg.	Ascorbic acid mg.	Vitamin D, I.U.
						I.U.	mg.				
Man (70 kg.)											
Sedentary	2,500	70–100	800	12	3,000	500	1·5	2·2	15	75	
Moderately active	3,000					600	1·8	2·7	18		
Active – very active	3,500–4,500					770	2·3	3·3	23		
Woman (56 kg.)											
Sedentary	2,100	60–90	800	12	3,000	400	1·2	1·8	12	70	
Moderately active	2,500					500	1·5	2·2	15		
Very active	3,000					600	1·8	2·7	18		
Pregnancy (latter half)	2,500	85	1,500	15	3,500	600	1·8	2·5	18	100	400–800
Lactation	3,000	100	2,000	15	5,000	770	2·3	3·0	23	150	400–800
Children up to 12 years											
Under 1 year	100*	4*	1,000	6	1,500	130	0·4	0·6	4	30	400–800
1– 3 years	1,200	50	1,000	7	2,000	200	0·6	0·9	6	35	
4– 6 ,,	1,600	60	1,000	8	2,000	270	0·8	1·2	8	50	
7– 9 ,,	2,000	70	1,000	10	2,500	330	1·0	1·5	10	60	
10–12 ,,	2,500	80	1,000	12	2,700	400	1·2	1·8	12	75	
Children over 12 years											
Girls 13–15 years	2,800	100	1,300	15	3,000	470	1·4	2·0	14	80	
16–20 ,,	2,500	100	1,000	15	3,000	400	1·2	1·8	12	80	
Boys 13–15 ,,	3,200	100	1,400	15	3,000	630	1·9	2·4	16	90	
16–20 ,,	3,800	120	1,400	15	3,500	770	2·3	3·0	20	100	

NOTES TO TABLE 1

(1) The values given represent the tentative goal towards which to aim in planning practical dietaries. It should be noted that the vitamin figures are calculated requirements for food as eaten and do not allow for losses in cooking. Since such losses may be extensive, especially in the case of the water-soluble vitamins, provision should be made for them in planning practical dietaries.

(2) For adults a higher range of protein requirements has been given than the minimum standard suggested by the League of Nations Technical Commission on Health. In practice it would be difficult to plan diets of normal composition, in the higher Calorie ranges at least, which contain as little as 1 gram of protein per kilogram of body weight.

The requirements for children have been calculated from the standards laid down by Sir John Orr (1939) and the table of average body weights of boys and girls.

(3) The requirements of vitamin A are stated in terms of International Units of pre-formed vitamin A, *i.e.* an allowance has been made for incomplete absorption of the part of the vitamin intake derived from carotene.

(4) The values given of vitamin C are those laid down by the Food and Nutrition Board, U.S. National Research Council. The League of Nations Technical Commission on Health standard for an adequate ascorbic acid intake for an adult is only 30 milligrams.

(5) Vitamin D is undoubtedly necessary for older children and adults. When not available from sunshine, it should be provided probably up to the minimum amounts recommended for infants.

(6) The needs of infants increase from month to month. The amounts of minerals and vitamins given are for approximately 6 to 8 months.

(7) Allowances for children are based on the middle year of each group (2, 5, 8, etc.) and for moderate activity.

TABLE 2

ESTIMATED ANNUAL CONSUMPTION PER HEAD OF CERTAIN FOODS IN THE UNITED KINGDOM AT THREE PERIODS

	1909 to 1913	1924 to 1928		1934	
		Quantity	As per cent of 1909 to 1913	Quantity	As per cent of 1909 to 1913
	lb.	lb.		lb.	
Fruit	61	91	149	115	188
Vegetables (other than potatoes)	60	78	130	98	164
Butter	16	16	100	25	157
Eggs (in numbers)	104	120	115	152	146
Cheese	7	9	128	10	143
Margarine	6	12	200	8	133
Sugar	79	87	110	94	119
Meat	135	134	99	143	106
Potatoes	208	194	93	210	101
Wheat flour	211	198	94	197	93

the various foodstuffs consumed per head in this country at three periods from 1909. It will be seen that there has occurred a substantial increase in the consumption of the so-called 'protective' foods, namely fruit, vegetables, butter, eggs and cheese, the annual consumption in 1934 being respectively 88, 64, 57, 46 and 43 per cent greater than in the period 1909 to 1913. Table 3 shows that the intake of protein and carbohydrate has remained essentially unaltered during the past thirty years. The consumption of fat, however, has increased in this period by 25 per cent. This is of considerable importance not only because of the high caloric value of fat but because foods rich in fats are very often carriers of the fat-soluble vitamins A and D, and in the case of milk and cheese are most valuable sources of the minerals calcium and phosphorus and of Vitamin A and riboflavin.

TABLE 3

FOOD CONSUMPTION IN TERMS OF CALORIES AND PROXIMATE PRINCIPLES

	Average 1909 to 1913	Average 1924 to 1928	Average 1934
	Per head per day	Per head per day	Per head per day
Animal protein, grams . .	43	43	46
Vegetable protein, grams .	43	42	41
Total protein, grams . .	86	85	87
Animal fat, grams . . .	87	91	109
Vegetable fat, grams . . .	12	19	15
Total fat, grams . . .	99	110	124
Carbohydrates, grams . .	436	431	425
Calories	3,057	3,139	3,246

THE NATION'S DIET TO-DAY

The statistics quoted in the last section, while clearly indicating an improvement in the national dietary, are only averages over the whole population. A more accurate picture of the state of affairs in Great Britain in the years before the outbreak of the Second World War in 1939 was revealed by the nation-wide dietetic survey undertaken by Sir John Orr and his fellow-workers at the Rowett Research Institute, Aberdeen. The results are fully set out in Sir John Orr's book entitled *Food, Health and Income*, and we are indebted to him for permission to publish the statistics and tables used in this chapter.

A brief summary of Sir John Orr's findings will serve to emphasize the serious state of under-nutrition of a section of the population in the period between the two world wars. The population was divided into six groups classified according to the income per head per week, as indicated in Table 4. In Table 5 is given the average composition of the diet in each of the six groups, along with the standard requirements per unit of population. If the diets of the different groups are compared with the standards shown in the right-hand column of Table 5, it will be seen that in Group 1, the lowest income level, where the weekly income per head is 10/- or less and the estimated average expenditure on food per head per week is only 4/-, the average diet is inadequate for perfect health in each of the fundamental nutritional requirements, namely energy value, protein, minerals and vitamins. Stated baldly, this means that when the survey was undertaken 10 per cent of the people of Britain were living in a state of severe under-nutrition.

TABLE 4

CLASSIFICATION OF THE POPULATION BY INCOME GROUPS AND AVERAGE FOOD EXPENDITURE PER HEAD IN EACH GROUP

Group	Income per head per week	Estimated Average Expenditure on Food	Estimated Population of Group	
			Numbers	Percentage
I	Up to 10/-	4/-	4,500,000	10
II	10/- to 15/-	6/-	9,000,000	20
III	15/- to 20/-	8/-	9,000,000	20
IV	20/- to 30/-	10/-	9,000,000	20
V	30/- to 45/-	12/-	9,000,000	20
VI	Over 45/-	14/-	4,500,000	10
Average .	30/-	9/-	—	—

Groups 2 and 3 obtain a diet which is adequate in energy-value and protein, but deficient in minerals and vitamins. It is not until Group 4 is reached, in which the weekly income is 20/- to 30/- per head, that the diet becomes adequate. The consumption of potatoes and flour is remarkably uniform in all groups, whereas the consumption of all other foods increases with a rise in income. Thus the total milk consumption rises from 1·8 pints per head per week in Group 1, to 5 pints in Groups 5 and 6. The amount spent on fruit rises from 2½d. per head per week in Group 1 to 1/8 per week in Group 6. The number of eggs eaten rises from just over one

TABLE 5

COMPOSITION OF THE DIET (PER DAY) BY INCOME GROUPS OF THE POPULATION

	Group 1		Group 2		Group 3		Group 4		Group 5		Group 6		Standard Requirements per unit of Population
Protein:	Grams	Per cent	Grams	Per cent	Grams	Per cent	Grams	Per cent	Grams	Per cent	Grams	Per cent	Grams
Plant	40·9	64·5	43·5	57·2	44·0	52·6	43·8	49·0	42·8	45·3	40·5	41·0	—
Animal	22·5	35·5	32·5	42·8	39·6	47·4	45·6	51·0	51·6	54·7	57·8	59·0	—
Total	63·4	100·0	76·0	100·0	83·6	100·0	89·4	100·0	94·4	100·0	98·3	100·0	68
Fat:													
Plant	20·9	29·2	17·9	18·1	14·5	13·2	13·3	11·0	12·2	9·4	11·1	7·9	—
Animal	50·7	70·8	80·9	81·9	95·1	86·8	107·3	89·0	118·3	90·6	130·4	92·1	—
Total	71·6	100·0	98·8	100·0	109·6	100·0	120·6	100·0	130·5	100·0	141·5	100·0	98
Carbohydrate	348	—	381	—	395	—	403	—	406	—	396	—	—
Minerals:													
Calcium	0·37	—	0·52	—	0·61	—	0·71	—	0·83	—	0·95	—	0·6[1] to 0·9[2]
Phosphorus	0·81	—	1·04	—	1·17	—	1·28	—	1·42	—	1·54	—	1·23
Iron	0·008	—	0·0099	—	0·011	—	0·012	—	0·0127	—	0·0137	—	0·0115
Vitamin A (Int. units)	774		1,250		1,624		2,015		2,210		2,875		1,900
Vitamin C (mg.)	42		57		66		79		92		116		70
Calories	2,317		2,768		2,962		3,119		3,249		3,326		2,810

[1] Minimum for positive balance.

[2] Minimum plus 50 per cent for safety margin.

egg a week in Group 1 to nearly five eggs a week in Group 6. The total fat consumption rises from 10·2 oz. per week in Group 1, to 15·8 oz. per week in Group 6; and the amount of animal protein from approximately 5 oz. per week in Group 1 to approximately 14 oz. per week in Group 6. It would appear, therefore, from a study of Table 5, that immediately before the War approximately half the population of Britain received a diet which fell short, in one or more respects, of the accepted nutritional standards for the maintenance of optimal health.

That the serious state of the nation's nutrition revealed by Sir John Orr had little or nothing to do with the fact that before the War Great Britain imported about two-thirds of its total food requirements is evident when these findings are compared with the state of nutrition over the same period in the United States of America, a country where the economic level is as high as, if not higher than, in Great Britain and which is a food-exporting country.

According to a recent publication (National Research Council Bulletin No. 109, *Inadequate Diets and Nutritional Deficiencies in the United States*, 1943), a survey of the diets of low-income families in several of the large cities of the United States over the same period as that covered by Sir John Orr's investigations in this country showed that 27 to 48 per cent of the families had an average daily caloric intake below that estimated to be the minimum requirement, while two-thirds of the families surveyed consumed diets at or below the minimum level for several protective foods. Conditions were a little better in rural areas, but even here anything from 10 to 30 per cent of families were found to be receiving poor diets which failed to meet the minimum requirements in one or more nutrients.

The minimum levels used as standards allowed no margin of safety, and no allowance was made for waste of food or loss of nutrients in preparation and cooking when calculating the nutritive value of the diets, so that the proportion of families with unsatisfactory diets was certainly much higher than that revealed by the surveys.

When compared with the optimum standards laid down by the Food and Nutrition Board of the National Research Council (see Table 1), fewer than one-fifth of the American families studied in the nation-wide surveys mentioned above were receiving diets which met the recommended allowances.

These disturbing facts about the inadequacy of the diets of large sections of the populations of two of the richest countries in the world shed some light on the magnitude of the problem confronting those responsible for carrying out the recommendations of the Hot Springs Conference of 1943. At this conference representatives of the United Nations agreed that the food resources of the world could and should be developed and allocated so that everyone, irrespective of his country, might receive a diet satisfying his nutritional requirements.

Any reader who feels that these aims are idealistic and visionary should turn to Chapter X where it is shown what can be achieved by planning the dietary on a national scale. The statistics quoted there show that the average war-time diet in Great Britain in 1943 was better than the pre-war diet in respect of almost all the essential nutrients. That this improvement was secured even under the stresses of war is not only a tribute to the success of those who controlled the national food supply, but also gives an inkling of how, in a world at peace, the resolutions of the Hot Springs Conference might well be fulfilled.

NUTRITION AT DIFFERENT INCOME LEVELS

In the present state of knowledge there is good evidence to suggest that the simple measurements of height and weight supply as much reliable evidence about the average state of nutrition of groups of individuals belonging to different strata of the population as can be obtained by any other means. If sufficiently large groups are examined, the effect of heredity is reduced to a minimum. No individual can pass the limit set by inheritance. The most that can be done by diet or other environmental factors is to enable the individual to attain his full inherited capacity for growth and health. The assumption that differences in stature between various economic classes is solely or even mainly due to hereditary factors cannot be accepted. Thus many observers in America have remarked on the extraordinary improvement in the physique of the children of Japanese, Russian and other immigrants to the United States, who within one generation are several inches taller than their parents. As H. B. Norman puts it, 'People on the same kind of diet tend to have the same stature, and the real correlation is often one of diet rather than of inherited genes.'

In addition to the factors of heredity and diet, the physical development of an individual may be adversely influenced by other conditions which are complex in character and whose relative importance is difficult to define and assess. Such factors are disease, poor housing, excessive fatigue, inadequate hours of sleep, ignorance of the principles of hygiene and nutrition, and lack of opportunities for relaxation and exercise in the fresh air. It will be admitted that persons belonging to the poorer classes are relatively at a disadvantage in all these respects, but, after making due allowances, the weight of evidence, as supplied from experiments and observations on both human beings and animals, strongly upholds the view that a diet inadequate quantitatively and qualitatively must be accepted as the most important single cause of defective growth and poor health. To test the correctness of this assertion it is necessary to compare persons belonging to the poorer classes (Groups 1 and 2) with the well-to-do (Groups 5 and 6) with respect to physique and the incidence and degree of ill health.

1. Children

Owing to increased nutritional requirements for growth, the effect of a faulty diet should be seen at its greatest in children and adolescents. Before the claims regarding the importance of diet can be accepted, it is necessary to show, first, that the children of the poor are smaller in size, lighter in weight and weaker in muscle and have a higher incidence of deficiency disease and a greater susceptibility to disease in general than children belonging to higher income groups; and secondly, that their development and health improve with an improvement in their diet.

Height and Weight. H. B. Norman (1939) carefully investigated the height and weight, and measured the muscular power by the spring-balance dynamometer, in 500 public school boys (Groups 5 and 6) and 500 secondary school boys in the County of Durham (Groups 2 and 3) and found that each year between the ages of 14 and 18 the public school boy was approximately 16 lb. heavier and 3 in. taller than the secondary school boy, and the power of his lumbar muscles was 30 lb. greater.

Deficiency Diseases. The tragic results of malnutrition were seen in Vienna, at the conclusion of the Great War 1914 to 1918, when that city became the centre for the study of every type and degree of deficiency disease, particularly in children. In general, however, the incidence of each condition has been greatly lessened by the dietetic improvements that have occurred in the past thirty years. For example, *rickets*, which has been predominantly a disease of the poor, is now only rarely seen in its grosser manifestations with marked skeletal deformities, but radiological investigation has shown that minor manifestations are more frequently encountered among the children of the lower-income groups.

Scurvy. This is another deficiency disease which was very frequent in the past both in children and adolescents. The fully developed disease is rare to-day, but the state of vitamin C nutrition, as judged by tests for the excretion of vitamin C in the urine, is less satisfactory among children of the poor than among those of the well-to-do.

Iron Deficiency Anæmia. This condition is exceedingly common among infants of the poor. A large-scale investigation carried out in Aberdeen by Davidson, Fullerton and Campbell (1935) showed that a subnormal blood level was present in 87 per cent of such infants, 15 per cent being severely anæmic.

Xerophthalmia. A striking example of the effect of poverty on nutrition and the incidence of deficiency disease was seen in Durham, where, in the economic depression of 1931, there occurred among children in the coalfield region several cases of the eye condition known as xerophthalmia, which is due to severe and prolonged deprivation of vitamin A, but which is practically never seen in this country under normal circumstances.

Teeth. It is common knowledge that the condition of the teeth in persons belonging to the poorer classes is markedly inferior to that of those in more comfortable circumstances. Although other factors like malocclusion and lack of cleanliness must be taken into consideration, there is a great deal of evidence to show that dental caries is related to some fault in the diet. The effect of change of diet has been observed in several primitive communities, such as the Eskimos and the inhabitants of Tristan da Cunha, whose members used to have remarkably sound teeth. When these communities come in contact with civilization and adopt some of the dietary habits of civilized people (usually the worst ones), the incidence of dental caries increases enormously.

Many dietary factors are involved in determining dental health and development; the parts played by calcium and phosphorus, and by vitamins A, C and D are discussed in Chapters IV and V. Since the final state of the teeth is determined during their period of growth, it is at least highly probable that the seeds of future dental disease are sown in infancy or even before birth.

Unfortunately the evidence as to the factor or factors responsible for the development of dental caries is conflicting; and out of this confusion three main theories have arisen, each with its own adherents:

- (i) Diets defective in calcium and vitamins A and/or D lead to poorly calcified and otherwise malformed teeth. That such diets favour the development of caries is evident from the fact that administration of calcium and vitamins A and D in adequate amounts has been shown to decrease the prevalence of hypoplastic teeth and caries among children.
- (ii) Caries is due to an excess of carbohydrate foods, especially of refined sugar.
- (iii) Soft diets which require little chewing and hence do not promote proper development of the jaws and teeth have also been implicated as a cause.

Contradictory evidence can be quoted for or against each theory and it is probable that more than one of the factors mentioned may be involved. It is sufficient to note that in its lack of calcium and vitamins A, C and D, and in its relative excess of carbohydrate food, the type of diet eaten by the lower-income groups in Britain before the outbreak of war in 1939 supported at least two of the above hypotheses. The extent of the problem will be realized from the estimate made about fifteen years ago that about 80 per cent of the deciduous teeth of British children showed hypoplasia as judged by surface structure, and that 90 per cent of the children had at least one carious tooth.

In a recent paper, M. Mellanby and Coumoulos (1944) have made a most interesting comparison between the state of dentition of 5-year-old London school-children in 1929 and 1943. That there has been a striking improvement in dentition is shown by the observations that in 1929 only 8 per cent of children had teeth of good structure, whereas in 1943 the percentage had increased to 19; and that in 1929 only 5 per cent of children were caries-free and 63 per cent had much caries, whereas in 1943 22 per cent were caries-free and only 29 per cent had much caries.

The teeth of the 1943 children were formed in the immediate pre-war years and the first year of the war, and it is difficult to avoid the conclusion that the improvement in dentition must have been due largely to the increased intake of milk and of vitamins A and D through the Government's policy of making free or cheap milk and cod-liver oil available to expectant and nursing mothers and their children. This subject is discussed in greater detail in Chapter X.

Infective Diseases. As in the case of the deficiency diseases, so in the infective diseases of childhood, it is well recognized that children belonging to the poorest classes succumb to infections such as pneumonia, tuberculosis and the exanthemata much more easily than do children of the well-to-do.

Defective Hearing. The relation of defective hearing to nutrition has been carefully studied by Kerridge, Briggs, Choyce and Hill (1939). The acuity of hearing was tested with a gramophone audiometer in 1,000 children living under good, and in 6,000 living under poor, social conditions. It was found that defective hearing was on the average about four times as common in the latter group as in the former. In the poorest places, whether urban or institutional, it may be ten times as common in a bad as in a good environment. Since it is generally agreed that 80 per cent of cases of defective hearing are due to infection of the middle ear, these findings strongly support the view that infections are much more frequent in the poor than in the well-to-do. An investigation of such environmental factors as climate, housing and the mixing of children showed that they had little effect on the incidence of defective hearing. On the other hand, careful studies proved conclusively that dietary deficiency was of the greatest importance. Thus, in orphanages in different parts of the country where the institutional accommodation and supervision of the children were similar, it was found that a far higher incidence of deafness, and therefore of infection of the middle ear, occurred in institutions in which the diet was badly balanced and deficient in the 'protective' foodstuffs. The authors conclude by saying that the most important work on the prevention of deafness will be done by those who are striving to improve the social conditions and, in particular, the nutrition of the nation.

The recent introduction of a system whereby milk in adequate quantities has been made available to school-children has given striking confirmation of the importance of the dietary factor in the diseases and states of subnormal nutrition discussed in the preceding paragraphs. Thus, in Scotland, in one experiment in which over 30,000 children on a poor home diet received a daily milk supplement, the rate of growth was more than 20 per cent greater than in those who were not given the supplement, and a notable improvement in health and vigour was observed.

2. Adults

The effect of dietary deficiency is not observed in children only. Where such deficiency persists, its results can be traced among the adult population. For instance, up to the last few years before the second world war two out of every three recruits presenting themselves to the army were rejected on the grounds of physical unfitness. The introduction of a scheme, however, whereby selected individuals from the rejected recruits were given an adequate diet and physical training for several months, showed that such measures improved the health to such an extent that a large proportion of the treated individuals could then be accepted for the army.

In 1939 compulsory service for young men of twenty was introduced in Great Britain. It was officially stated that approximately 90 per cent of the militiamen presenting themselves for medical examination passed the accepted standard of physical fitness. To attribute this remarkable improvement on the previous figures solely to changes in the nation's diet would be unwise. For one thing it is difficult to compare the health of the militiamen drawn from all classes with recruits for the regular army, who were mainly drawn from the poorer sections of society. Hurried medical examination in a time of national emergency was also a factor tending to exaggerate the high percentage of militiamen graded as A1; and in addition, the authorities may have deliberately accepted some individuals slightly below the required standard, since they possessed the knowledge, derived from the experiment just mentioned, that a marked gain in physique could be expected to accrue as a result of good food and exercise in the fresh air. Nevertheless, it is justifiable to assume that a wonderful change for the better in the health and physique of the present generation of young men has actually occurred during the past ten years, during which period intense propaganda and effort have been undertaken to improve the nation's diet.

Hypochromic Anæmia. Probably the most common deficiency disease in adults is the chronic nutritional hypochromic anæmia of women. This is essentially a disease of the poor and is due to the prolonged intake of a diet deficient in iron. Of some 1,750 women belonging to the lower-income groups examined in Aberdeen by Davidson, Fullerton and Campbell nearly 50 per cent were shown to be suffering from some degree of anæmia.

Infective Diseases. Dietary deficiency reduces the resistance to infective diseases in adult life just as it does in childhood. For example, the Registrar-General's Report for 1927 showed that the mortality rate for tuberculosis among occupied males was nearly three times higher among unskilled labourers than among the higher ranks of business and professional life.

Physical Characteristics. The factors influencing nutrition among individuals of different income levels in this country also play their part in determining the physical characteristics of the different races and tribes of men. A most instructive example of a natural nutritional experiment was revealed by investigations in Africa into the physical attributes and health of two neighbouring tribes of Kenya natives, the Masai and the Kikuyu, who live under the same climatic and housing conditions but on different diets. The diet of the Masai is rich in milk and meat and contains abundant first-class protein, minerals and vitamins, while the diet of the Kikuyu is mainly a cereal one, therefore rich in carbohydrate but poor in other constituents, and corresponds roughly to the diet eaten by Groups 1 and 2 in this country (see Table 5). The males of the Masai tribe are on the average no less than 5 inches taller than the males of the Kikuyu tribe, while bodily deformities, dental caries, and pulmonary and intestinal diseases are more than twice as prevalent among the Kikuyu.

Experimental Studies. Another method of investigating the effects of diet on growth and health is that of animal experiment. It is only necessary to refer to the work of Major-General Sir R. McCarrison who made a lifelong study of nutritional problems in India. Groups of rats belonging to the same families were fed on diets common to the various races in India. The rats were kept under the same environmental conditions. The physique and the incidence of disease in each of these groups corresponded to an astonishing degree with those of the races whose diets were copied. A similar experiment was carried out in Aberdeen: rats were placed on the diet of the poor, as estimated in Peterhead and Aberdeen, and control groups were given the same diet, plus milk and green vegetables. It was shown that not only was the rate of growth of the control animals on the good diet much greater and their fertility higher, but that their resistance to disease was superior. A spontaneous epidemic infection in the animal house in which both groups of rats lived produced a death rate of 11·6 per cent in the animals on the good diet compared with 54·3 per cent in the animals on the poor diet.

Confirmatory Data. The data collected and set forth by Sir John Orr were amply confirmed by the late G. C. M. M'Gonigle, who, as Medical Officer of Health for Stockton-on-Tees, conducted intensive investigations into the relation between nutrition and the health of the population which was under his supervision. With Kirby he summed up the results of this work in *Poverty and Public*

Health (1936), in which the view is expressed that, of the three main factors responsible for man's health or lack of it – namely, nutrition, infection and external environment – the first of these, nutrition, is the most important. Proof of the paramount importance of nutrition is advanced in the results of a study of the health of a large group of people transferred from an overcrowded slum to a municipal housing estate. Contrary to expectation, there was a significant increase in the death rate. This is explained as being due to the increased rent which had to be paid; as a result, the expenditure on foodstuffs was even less than before, and dietary deficiencies were enhanced. This experience supports the statement already made that the nutritional problem at the present day is mainly a social and economic one.

As a final example of the recognition of the urgent need for more adequate nutrition of the population in the interests of health, reference may be made to the *Medical Testament* issued by the Cheshire Panel Committee and published in the *British Medical Journal* Supplement of April 15th, 1939. This manifesto avers that, while the medical services rendered available to the public by the National Health Insurance Act have increased the expectation of life, the promise of the Bill with regard to the prevention of sickness has not been fulfilled. According to the members of the Cheshire Panel Committee, their daily work among the sick has convinced them that much of the illness with which they have to deal results from a lifetime of wrong nutrition. In their own words, 'No health campaign can succeed unless the materials of which the bodies are built are sound. At present they are not. Probably half our work is wasted, since our patients are so fed from the cradle, indeed before the cradle, that they are certain contributions to a C3 nation. . . . Against this the efforts of the doctor resemble those of Sisyphus.'

The statistics quoted in the preceding sections are sufficient to show the magnitude of the nutritional problem facing this country to-day. The report of the British Medical Association's Committee on Nutrition (1933) gave 6/4 (at the then prevailing prices) as the minimum weekly expenditure on food per head necessary to maintain health, and a glance at Table 4 will show that, prior to 1939, 30 per cent of the population of this country spent less than this. While it is certain that nothing but an increase in income could raise the diets of the majority of this group to the standards of adequacy, there is no doubt that much could be achieved by a wider dissemination of the elementary facts of nutrition. Every effort should be made to increase the consumption of the so-called protective foods, and it is on this that the emphasis must at all times be laid. The wholesale and indiscriminate distribution of vitamin supplements to any section of the population in an attempt to improve the diet is quite unwarrantable and ineffective, as has been shown by several large-scale experiments. Vitamin preparations have their place in dietetics, but should only be given under certain specific conditions—*e.g.*, vitamin D in pregnancy, infancy, and childhood, or vitamin therapy where there is clear evidence that a vitamin deficiency exists or is impending. Under all other conditions the provision of a properly balanced diet will easily cover the vitamin requirements. A continuous drive is required to educate the people in regard to the best methods of cooking and preparing food for consumption, and to advise on the cost of foodstuffs relative to their nutritive value. It is with this object that the authors have devoted the next part of this book to a simple account of the physiology of nutrition.

PART II

THE PHYSIOLOGY OF NUTRITION

CHAPTER II

THE ENERGY REQUIREMENT

FOOD is required by the organism for the production of energy, to replace the tissues lost by wear and tear and to promote growth.

It has been shown that certain of the vitamins and essential amino-acids have among their functions that of regulating the processes involved in the production of energy and the promotion of growth and repair. Therefore, to the requirements stated above, we should for completeness add yet another: namely, food must supply the substances which control the functions of energy production, growth and repair.

For the adequate fulfilment of these purposes the diet must supply the following factors in sufficient quantity: 1. Calories; 2. protein; 3. vitamins; 4. minerals; and 5. water. With a few exceptions, such as the restriction of the energy intake in the treatment of obesity, the fulfilment of the above criteria is essential in the prescription of any diet. It is therefore necessary to examine these fundamental factors in detail.

1. The Calorie Requirement

A warm-blooded animal expends energy:

1. In maintaining the body temperature.
2. In promoting the activity of the vital organs.
3. In the performance of mechanical work.

The energy expended in the first two activities is called the *basal metabolism*, which may therefore be defined as the lowest expenditure of energy consistent with a continuance of the vital processes. This minimal or basal level is secured when the body is completely at rest in a normal environmental temperature and fasting. Under these conditions heat production, whether by mechanical work or by the activity of the various organs, is at a minimum, and the metabolism is not being influenced by the stimulus which it receives from foods – the so-called specific dynamic action of the foodstuffs.

The total energy expenditure of the body is the sum of the basal metabolic requirement, the energy expended as a result of the specific dynamic action of the foodstuffs and the energy required for the performance of mechanical work. The energy output is always expressed as units of heat, the unit used being the large Calorie, which is defined as the amount of heat required to raise the temperature of a litre of water from 15° to 16° Centigrade.

In the conversion of energy from heat to mechanical work, no fuel-driven machine is completely efficient, and a large proportion of the heat derived from the burning fuel has to be wasted. The human body is no exception to this rule. The amount of food material that is burned to perform mechanical work produces many times the amount of energy which appears as mechanical work, the excess energy appearing as heat. Therefore, when we refer to the energy required for the performance of mechanical work, we mean, of course, the sum of the mechanical work plus the heat produced in its performance.

The heat developed when mechanical work is performed can be used in maintaining the temperature of the body. But unless the environmental temperature is low, the heat produced under basal conditions, when the amount of muscular activity is small, is more than adequate to maintain the body temperature, owing to the insulating effect of the clothing. Therefore, unless the temperature of the air is low, the extra heat developed when mechanical work is done represents waste that must be got rid of if the body temperature is to be kept at a normal level.

2. The Basal Metabolic Requirement

When it is necessary to compute this accurately, the method of indirect calorimetry may be used, in which the oxygen consumption of the individual is measured under basal conditions by collecting and analysing the expired air. The heat production is calculated from this. The basal energy output of an individual is determined by some fundamental condition of bodily nutrition such as the active mass of body protoplasm. It is not proportional to the body weight, but is more nearly proportional to the surface area of the body than to any other measurable factor. The basal energy output is also influenced by age and sex, so that in accurate estimations of the basal metabolism, the age, sex, and surface area of the individual must all be taken into account.

In estimating the dietary requirements, such accuracy is not essential except under experimental conditions, and a sufficiently close approximation can be reached on the basis of body weight, the basal energy requirement of an adult during 24 hours being reckoned as 11 Calories per pound of body weight (roughly 25 Calories per kilogram). It must be emphasized here that the term 'body weight' used in this computation refers to the *ideal* body weight corresponding to the age, height and sex of the individual (see Table 17).

The high maintenance costs of the human machine are not always realized. Some 50 per cent of the total daily energy expenditure of a man undertaking moderate muscular work for some hours during the day goes to satisfy the basal metabolic requirements; and the proportion falls only to about 40 per cent even when hard muscular work has to be undertaken.

3. The Specific Dynamic Action of the Foodstuffs

If food is taken by an individual at rest, there is an increase in heat production over the basal level. The amount of this increase varies according to the nature of the food taken. Thus:

Protein	increases	metabolism	by 30	per cent
Fat	,,	,,	4	,,
Carbohydrate	,,	,,	5	,,

This means that if a starving individual kept at complete rest in a normal environmental temperature is given protein in an amount which should meet the basal caloric output, his output of energy will now increase by 30 per cent, whereas with a similar amount of fat it would only increase by 4·1 per cent, and in the case of carbohydrate, 4·9 per cent.

The cause of the 'specific dynamic action' of the foodstuffs or, as it has been more recently termed, the 'calorigenic effect', is not definitely known, but there seems to be a stimulation of the cells of the body to greater activity by certain constituents of the foodstuffs after their absorption. In the case of protein, the specific dynamic action can be attributed to the metabolism of the amino-acids liberated from protein. Individual amino-acids have been shown to exert specific dynamic actions of varying intensity, and the total specific dynamic action of a protein is the sum of the specific dynamic actions of its constituent amino-acids. When protein is being used to repair tissue wear and tear, however, it exerts no specific dynamic action, and it is therefore only in the exogenous metabolism of the amino-acids that the specific dynamic action of protein appears.

It is important to realize that the energy liberated by the specific dynamic action of protein cannot be used in the performance of mechanical work. Accordingly when drawing up a table of the energy requirements, allowance must be made for the energy expenditure due to specific dynamic action over and above the amount necessary to satisfy the basal and work requirements; an increase of 5 to 10 per cent is usually allowed for this purpose.

The heat produced by the specific dynamic action of protein can, however, be used to maintain body temperature. This is a matter of importance to inhabitants of cold regions who, through force of circumstances, have to subsist on a very high protein diet.

Conversely the specific dynamic action of food is at its highest when the environmental temperature is high. This has been used as an argument that the protein intake should be limited in hot climates; but the importance of this effect of protein has been somewhat exaggerated, since the extra heat which is produced is only a very small fraction of that resulting from muscular work. In practice, a high protein diet has no demonstrable deleterious effect on the capacity for work at high temperatures, and it is therefore unnecessary to restrict the protein intake in hot climates below the normal quota.

4. The Energy Expenditure for Exercise and Work

Only in accurate studies is it necessary to make exact calculations of the total energy requirements. For ordinary purposes, it is sufficient to adopt a round figure for the individual's total energy requirements and prescribe accordingly. The figure usually taken as fulfilling the total energy requirement over 24 hours in an adult who is not engaged in heavy muscular work is 16 Calories per pound of ideal body weight (roughly 35 Calories per kilogram). The usually accepted figures for the total energy expenditure over 24 hours in a man weighing 11 stones (70 kilograms) are:

2,500 . . .	Calories for	sedentary occupation	
2,500 to 3,000 . .	,, ,,	light muscular work	
3,000 to 3,500 . .	,, ,,	medium	,, ,,
3,500 and upwards .	,, ,,	heavy	,, ,,

Men engaged in heavy occupations (lumbermen, blacksmiths) may consume 5,000 Calories daily, while the caloric intake of some athletes at the Olympic Games of 1936 was estimated at approximately 7,000.

The figures for the Calorie requirements given above will doubtless raise in the reader's mind the question of how it has been possible for whole communities to exist on much lower food intakes, as for instance in Central Europe in the first World War or in the large parts of German-occupied Europe in the second, without most of the members dying from gradual starvation.

Experimental observations have supplied the answer to this question. They have shown that if healthy adults are placed on a diet as low as 2,000 Calories but adequate in other respects, and if on this diet they carry out their usual activities, the body weight at first falls rapidly but later becomes steady at a level about 10 per cent below normal. The maintenance of body weight (after the initial fall) on a theoretically inadequate Calorie intake is secured by a reduction in the total energy expenditure—*i.e.* the basal metabolism falls steadily with the weight until it becomes stationary at a level of anything up to 25 per cent below normal, while the energy expended in work is lessened both by a reduction in the amount of energy used in carrying out any given activity such as walking and by a reduction in the actual amount of work done during the day.

It is probable that long-continued existence on such a meagre diet produces undesirable effects other than a reduced capacity for strenuous physical work; resistance to infection, for instance, is almost certainly decreased. But experience, derived chiefly from the treatment of diabetic patients, has shown that normal activity and only a slightly subnormal body weight can be maintained on diets providing less than the ideal number of Calories for varying activities enumerated above. Thus, an intake of 2,000 to 2,500 Calories or even less is usually found to be adequate for diabetics in sedentary occupations, while many diabetics engaged in labouring jobs maintain good health on 3,000 Calories daily. The total energy requirements of women, owing to their lower body weight, are less than those of men but their requirements per pound of body weight are the same. The energy requirements of children are discussed in the section dealing with diet in childhood (see p. 200).

The Relative Value of the Foodstuffs in meeting the Energy Requirements. The amount of heat given out by the three classes of foodstuff when burned in the body (after allowance has been made for loss through non-absorption) has been shown to be as follows:

1 oz. of protein yields	114 Calories	(1 gram protein = 4 Calories)
1 oz. of fat yields	256 Calories	(1 gram fat = 9 „)
1 oz. of carbohydrate yields	114 Calories	(1 gram carbohydrate = 4 „).

Theoretically, any of the three classes of foodstuff could supply the energy requirements of the body, but several fundamental physiological facts make it essential that an adequate diet should contain all three. Of these perhaps the most outstanding is the need for the maintenance of *nitrogenous equilibrium.* In starvation, a constant breakdown of body protein occurs and a negative nitrogen balance results, since the nitrogen lost in the excreta (which represents the end-products of the protein continually being broken down in the body) is not replaced by ingested protein. For the replacement of the body protein lost in the wear and tear of the tissues an adequate supply of protein in the diet is essential. One of the most important requisites of an adequate maintenance diet for adults is that it should keep the individual in nitrogenous equilibrium, that is, the amount of nitrogen in the food should at least equal that lost in the excreta. In children, in whom protein is required for the growth of the body tissues, the nitrogen intake must exceed the loss.

Special Value of Carbohydrate. The protein of the diet will only replace body protein lost in the wear and tear of the tissues when sufficient carbohydrate is ingested simultaneously to maintain energy equilibrium. In the absence of sufficient carbohydrate for this purpose, ingested protein, instead of being used to repair tissue wear and tear, is burned as fuel. The amount of protein required to maintain nitrogenous equilibrium is thus greatly reduced by the ingestion of carbohydrate at the same time – in other words, by the so-called *protein-sparing action* of carbohydrate. According to Cuthbertson and Munro (1939), to yield this protein-sparing action the carbohydrate must actually be eaten at the same meal as the protein. They found, in experiments on human subjects, that if the protein and fat of the day's diet were eaten at a different meal from the carbohydrate, the subjects excreted more nitrogen than they ingested in the day by an amount about equal to that lost from the body in protein starvation. The ingested protein was therefore being used as fuel and not in repairing tissue, and this suggests that dietary protein divorced from carbohydrate is of no use in repairing the wear and tear of body protein.

Carbohydrate is essential for life, and during starvation protein is broken down to produce glucose in order to keep the blood sugar at a constant level. A certain minimum amount of carbohydrate must also be metabolized to ensure the complete oxidation of fat; otherwise ketosis will develop (see p. 234). Apart from these considerations, carbohydrate has other advantages as a part of the diet. It is the most readily available source of energy for muscular activity, and carbohydrate foods are cheap. A diet of which about half the total number of Calories are derived from carbohydrate is most suitable as regards protein-sparing action and bulk.

Special Value of Fat. Fat is important in that it has, weight for weight, over twice the caloric value of carbohydrate or protein, and so forms a concentrated source of energy. This is of great importance, since a diet in which the caloric requirements are supplied exclusively by protein and carbohydrate is excessively bulky. Moreover, at least two of the essential vitamins occur in association with natural fat, which contains in addition certain unsaturated fatty acids (*e.g.* linoleic acid) essential in small amounts for some animal species.

CHAPTER III

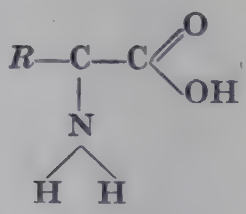
THE PROTEIN REQUIREMENT

In the discussion of the energy requirements of the body in the previous chapter, reference was made to the fact that protein has a far more important part to play in the diet than as a source of energy; it is therefore necessary to study in some detail both the significance and the requirements of protein in the diet.

Composition and Biological Value of Proteins

Proteins are the most important substances in the organic kingdom. They are normal constituents of all animal cells, being essential components of both protoplasm and nucleus. Further, many of the substances connected with vital processes – enzymes, hormones, and immune bodies – are either proteins or protein derivatives. In addition, proteins play an important part in regulating the osmotic relations between the tissue cells and the surrounding body fluids.

Proteins occur as very large molecules, which can, however, be broken down by hydrolysis into a number of simpler units – *the amino-acids.* These are all characterized by the presence of a carboxyl (COOH) group, with acidic properties, and an amino (NH2) group, with basic properties, attached to the same carbon atom. The rest of the amino-acid molecule varies with the particular amino-acid, and the structure of any amino-acid may be represented by the formula:



where *R* represents the remainder of the amino-acid molecule.

Over twenty of these amino-acids exist in nature. In the protein molecule they are linked together by the peptide linkage, in which the basic (amino) group of one amino-acid is linked to the acidic (carboxyl) group of the other acid, with the elimination of a molecule of water. Since any two amino-acids can be joined to form two different peptides by the linkage described above, and since over twenty amino-acids are found in the protein molecule in varying amounts in the different proteins, it is obvious that the [illegible] no matter how com-

possible num[illegible] proteins which may exist in nature [illegible] If, infinite. Th[illegible]eins of each species are characteristic of th[illegible] species – t[illegible]ein of beef muscle, for instance, differs from that of pork m[illegible] It is to this specificity of proteins that the phenomenon o[illegible]y and anaphylaxis is attributable.

In th[illegible]s of digestion, the large protein molecules are broken down [illegible]r constituent amino-acids, which are non-specific in the b[illegible] sense. The absorbed amino-acids are distributed to the [illegible] the blood stream. In the tissue cells, the amino-acids are [illegible]ombined with each other to form the protein charac[illegible] the particular tissue or utilized to form some of the o[illegible]ances essential to the functioning of the body, such as [illegible]r hormones. After the needs of the body for these two [illegible]have been met, an excess of amino-acids may still remain. [illegible]ve their amino-group split off in the process known as [illegible]tion, and the remainder of the molecule is then utilized [illegible]rce of energy by oxidation to CO_2 and water, either directly [illegible]er prior conversion to glucose or fatty acid.

[illegible]is usually accepted that consumption of protein in excess of [illegible]requirements over prolonged periods is of no permanent value [illegible]the adult, since, unlike carbohydrate and fat, neither proteins [illegible]r amino-acids are stored as reserves, nitrogenous equilibrium [illegible]ing rapidly established at a higher level when increased amounts [illegible]f dietary protein are taken.

Experiments on the regeneration of plasma proteins after repeated bleeding have, however, suggested that there exists in the body a labile reserve of body protein, which is probably represented by that part of the protein of organs and tissues which can be given up without loss of function.

The Essential Amino-acids

Plants can synthesize all the amino-acids they need from simple chemical compounds, but animals cannot do this, and to obtain the amino-acids for building protein they must therefore prey upon plants or upon animals which have in their turn preyed upon plants.

We have already said that natural proteins contain over twenty amino-acids, and the question naturally arises whether all of these are essential food constituents.

This question was answered, at least so far as the rat is concerned, when it was shown that young [illegible]s grew normally on a diet in which a mixture [illegible]ure amino-acid[illegible]med the only source of [illegible] however, removal of certain of the individual amino-acids from the mixture produced either loss of weight, and ultimately death, or in some cases failure of growth only, removal of others had no effect on health or on the rate of growth. As a result of this work it is possible to enumerate ten amino-acids which are essential for the maintenance of health and normal growth in the rat: namely, phenylalanine, lysine, tryptophane, methionine, valine, leucine, isoleucine, histidine, arginine and threonine. It follows that the rat must be able to synthesize the non-essential from the essential amino-acids.

There are undoubtedly species differences in the amino-acids which are essential for growth: for instance, glycine can be synthesized by the rat but is an indispensable amino-acid for the chick. Such experiments as have been carried out in men suggest that, with the possible exception of histidine, the same ten amino-acids are essential for the maintenance of health and growth in man as in the rat. Furthermore, it is possible to maintain healthy young adult males in nitrogen balance on a nitrogen intake of about 7 grams per day, of which 95 per cent is in the form of the ten essential amino-acids mentioned above; so that man also appears to be able to synthesize the non-essential amino-acids from the essential ones.

Though not yet definitely established, it has been suggested that the essential amino-acids are required for other vital functions, as well as the construction of new protoplasm. For example, methionine is required for the maintenance of normal liver function; phenylalanine is a precursor of adrenalin, thyroxin and melanin; and histidine is a precursor of purines.

The Biological Value of Proteins

Enough has been said to show that the essential food substan are not the proteins themselves but their constituent amino-aci Ideally, therefore, the adequacy of a diet in respect of prot should be considered in terms of its content of amino-acids rat than of protein; this would of course be a complicated proced and at any rate is not yet possible since knowledge of the am acid content of the different proteins and of man's requiremen the different amino-acids is as yet incomplete.

The nutritive value of a protein depends on the relation of essential amino-acids in its molecule to those required for building of new tissue a[illegible] essential substances. If the protei the diet is deficient in one or more of the essential amino-aci nitrogenous equilibrium cannot be obtained, no matter how complete and excellent the diet may be in all other respects. If, however, another protein containing the missing amino-acid in adequate amounts is added to the diet, nitrogenous equilibrium and normal nutrition can be established. This capacity of proteins to make good one another's deficiencies is known as their *supplementary value.*

The relative importance of the different proteins in nutrition is expressed in what is known as their *biological value* – *i.e.* the completeness with which they supply the essential amino-acids. The biological value of proteins has been estimated by animal experiments in two main ways. In the nitrogen balance method, the biological value is expressed as the percentage of the absorbed dietary nitrogen that is retained when the protein under investigation forms the only source of nitrogen in the diet. The growth method consists in determining the amount of the particular protein to be added to a protein-free diet in order to secure optimal growth.

As a result of these experiments it is possible to divide proteins into two main groups: those of high and those of low biological value. As might be expected, the proteins of animal origin are of high biological value and are hence spoken of as first-class proteins, while proteins of plant origin are generally of low biological value. Among the first-class proteins, egg and milk proteins have the highest biological value; next come the proteins of certain organs (liver and kidney and heart); and finally the proteins of meat and fish muscle and of the remaining organs.

A notable exception to this rule is gelatin, a protein derived from the collagen of connective tissue by boiling. Gelatin is entirely lacking in at least two essential amino-acids and is deficient in certain of the others, and therefore would not support life if it were the only source of amino-acids in the diet. In this sense its biological value is nil. Nevertheless, gelatin possesses some nutritive value as a supplementary protein (*e.g.* it is relatively rich in lysine), but its main value in the diet is as a culinary adjunct.

Among the plant proteins, potato protein has a biological value not much below that of meat, cereals are somewhat lower, then come the legumes and the nut proteins. The low biological value of plant proteins should not be taken as meaning that they form an unimportant part of the dietary. Theoretically, it would be possible to meet the complete needs of the body for amino-acids from a suitable mixture of plant proteins, but this is of little practical importance in human nutrition owing, first, to the inherent difficulties in obtaining the correct mixture of vegetable protein, and secondly, to the bulkiness of the diet which would be needed to compensate for the low protein content of most plant foods. But we have seen already that a protein deficient in one or more of the essential amino-acids can be made dietetically adequate by the addition of another protein supplying the deficient amino-acid in sufficient amount. Plant proteins can be supplemented by relatively small amounts of first-class protein so as to provide completely satisfactory diets from the point of view of amino-acid intake. It has in fact been shown by experiment that a mixture of plant and animal protein, where the latter forms less than half the total amount, has almost as high a biological value as that of the milk proteins.

In the war-time dietary of Great Britain, the average protein intake per head in 1943 was 87 grams, of which only 40 grams was of animal origin. The remaining 47 grams of protein was derived chiefly from bread, etc., made from flour of 85 per cent extraction, and potatoes – foodstuffs which, along with the legumes, form valuable sources of protein, especially when the consumption of meatstuffs has to be curtailed.

Dietary Requirements of Protein

As we have pointed out already, protein requirements should ideally be considered in terms of amino-acids, but in the present state of knowledge we must confine ourselves to discussing the dietary intake in terms of protein itself.

We have seen that protein is essential for the construction of new tissues and of substances like enzymes and hormones which are necessary for the functioning of the body.

The requirements for the construction of new tissue will obviously be greater in the young or in patients recovering from wasting diseases, but even in normal adults there is need of protein for the repair of the constant wear and tear of body protein, the occurrence of which finds expression in the small but constant excretion of urinary nitrogen in starvation or in subsistence on a protein-free diet. Recent feeding experiments with amino-acids containing isotopic nitrogen (radio-active nitrogen, N^{15}) in their amino groups have shown that the repair of body protein 'wear and tear' does not involve the replacement of a small number of the protein molecules of the body by new protein molecules made from the amino-acids absorbed from the food. On the contrary, a rapid and continuous regeneration of all the body proteins is constantly occurring, and there is a continual interchange of whole amino-acids or amino-groups between the proteins of the body and the amino-acids of the food circulating in the body fluids. The greater proportion of the amino-acids absorbed from the food takes part in these reactions, and only a small fraction is treated as surplus and deaminated without being built into protein molecules.

Much study has been devoted to determining the essential or optimal amount of protein in the diet; and it is obvious that in discussing this question we must bear in mind both the biological value of the proteins and the possible significance of the continual state of flux that has been shown to occur among the protein molecules of the body.

On a protein-free diet of adequate energy value, a normal man has an endogenous nitrogen excretion corresponding to about 20 grams of protein a day. Many workers have shown that it is possible to maintain nitrogenous equilibrium over long periods on diets containing little more protein than this. The lowest recorded intake known to us is 25 grams of protein per day, on which a Swiss doctor named Rose claims to have maintained nitrogenous equilibrium for four years, while a recent report, also from Switzerland, makes similar claims for diets containing 30 to 35 grams of protein. Other workers have found that health declined on such low intakes of protein, which is not surprising when one considers the bizarre nature of the diets required to ensure such a low protein intake, and the possibility that the intake of other essential nutrients may have been deficient.

These experiments certainly show that while there are obviously individual variations, it is possible to maintain nitrogenous equilibrium on an intake of protein not much greater than that corresponding to the endogenous protein metabolism which continues on a protein-free diet. Unlike carbohydrate and fat, protein and amino-acids are not stored; and if in a normal adult the protein intake is increased above the minimal level, there is a rapid rise in the urinary excretion of nitrogen, and nitrogenous equilibrium is established again at a higher level. Although it is certain that nitrogenous equilibrium can be well maintained on an intake of 50 grams daily, it is to be noted that the protein intake of the average normal adult male European whose diet is not restricted by economic conditions is in the neighbourhood of 100 grams daily, *i.e.* over twice the minimum requirement.

This raises two contrasting questions:

(*a*) Is a protein intake above the minimum necessary, since it does not lead to protein storage? And if not, does a protein intake much in excess of the minimum requirement produce any harmful effects?

(*b*) Does a protein intake well above the minimum promote vigour and physical efficiency?

The American physiologist Chittenden attempted to answer these questions by keeping healthy adult males, including himself, on diets containing as little as 40 grams of protein daily for periods of up to one year. Chittenden maintained that this procedure not only maintained but often increased physical and mental vigour.

Chittenden and other physiologists have argued from these results that reduction of the protein intake to the low levels used in his experiments is actually beneficial, claiming that the extra work done by the kidneys in excreting the nitrogen from intakes of protein higher than this throws a strain on the kidneys and tends to cause renal and vascular disease.

Animal experiments designed to support this theory have given inconclusive results, and all the evidence from human dietaries suggests that the kidneys can cope with the excretion of the nitrogenous end-products of large protein intakes without suffering damage as a result. For example, the incidence of renal and vascular disease among the Eskimos, who live on a diet largely composed of meat and consume two or three times as much protein as the average European, is no greater than in civilized communities. The Australian range-rider, the gaucho of the South American plains, and the Masai warrior of Central Africa all build up and maintain their superb physique on diets which contain anything up to 250 to 300 grams of protein per day.

It is therefore probably safe to say that provided the diet is properly balanced, particularly in respect of minerals and vitamins, a high protein intake can be tolerated throughout a lifetime without ill effects.

In the opposite camp to Chittenden and his school there are several advocates of a high protein diet who would give an affirmative answer to the question 'Does a protein intake well above the minimum promote vigour and physical efficiency?' and who maintain that rigid protein restriction over long periods may actually be harmful. These views are founded chiefly on breeding experiments on animals which show that lowered general resistance and early senility occur in successive generations when the protein content of their diet is quantitatively or qualitatively defective.

If the dietaries of various races throughout the world are studied, we find that there is a great latitude in the amount of protein on which man can subsist. The diet of the Eskimos, as we have seen, contains large amounts of protein, which supplies about 40 per cent of the total caloric intake, while the protein in the diet of the rice eaters of Java provides less than 10 per cent of the total Calories. Observations on the effect of diet on racial constitution are often quoted in support of the view that a generous intake of protein is desirable – as for example the contrast between the poor physique of the Bengalis as compared with the fine physique of the Sikhs. While it is true that the diet of the Sikhs contains more meat and milk, and thus more protein than that of the Bengalis, there are many deficiencies other than of protein in the Bengali diet, and it is therefore not justifiable to attribute the difference in physique between the two races solely to the different levels of protein intake. In general, however, the protein-eating races are superior in build and vigour to the cereal-eating races.

Another consideration to be taken into account in any discussion of protein requirements is the recent evidence, discussed on p. 28, that there is a constant interchange going on between the amino-acids of the food and those in the protein molecules of the body, almost all the absorbed amino-acids taking part in this process. Reduction in the amount of dietary protein will obviously tend to reduce the number of these interchanges; and while it cannot yet be stated that such reduction is harmful, it seems reasonable to assume that the continuous chemical activity of the body proteins is a vital process of fundamental significance, and accordingly that a sufficiency of amino-acids for this purpose should be provided by the diet.

The evidence as it stands is therefore in favour of a generous protein intake, and Sherman has summed up the arguments by setting the standard allowance of an adult at 1 gram of protein per kilogram of body weight (*i.e.* roughly 0·5 gram per pound), which provides a margin of safety of up to 100 per cent over the minimum required to maintain nitrogenous equilibrium. This standard has been adopted by the League of Nations Report on the Physiological Basis of Nutrition (1935) and more recently by other authoritative bodies.

It is impossible to consider the quantitative aspects of the protein requirement without taking into account the quality of the protein. In the standard mentioned above, it is assumed that the protein will be derived from a wide variety of foodstuffs of both animal and plant origin; or as the League of Nations Report puts it, 'the protein should be derived from a variety of sources and it is desirable that a part should be from animal sources'. It is difficult, and indeed impossible, to give a precise figure for the proportion of the protein intake which should be of a high biological value – indeed, nitrogenous equilibrium has been maintained, over short periods at least, on a total protein intake of only 50 grams per day, of which not more than 5 grams was of animal origin. But it has been generally agreed that, of the standard allowance of 1 gram protein per kilogram body weight, about 50 per cent should be protein of high biological value – *i.e.* about 35 to 40 grams for an average adult male. In persons whose diet is unrestricted by economic circumstances, the proportion is usually higher. *It is not generally realized that an average helping of meat* (3½ *oz.*) *at one meal, plus half a pint of milk daily will supply about* 35 *grams of first-class protein, and that cereals and vegetables will supply another* 50 *grams of protein of lower biological value.*

In dealing with the protein requirement, it must be emphasized that if a sufficiency of fat and especially of carbohydrate is provided in the diet, muscular work does not increase the rate of protein breakdown – in other words, although protein can be used as fuel, it is not *needed* as such. The protein requirement of the clerk is therefore as great as that of the navvy, and the increased amount of protein foods usually eaten by working men is not an expression of a strict physiological requirement but is due rather to the fact that the appetizing protein foods are needed to increase the palatability of the large amounts of carbohydrate and fat foods which must be eaten to satisfy the energy expenditure involved in hard muscular work.

Protein Deficiency

An inadequate protein intake is known to limit growth, impair fertility and diminish physical and mental vigour, but there is no specific clinical syndrome formed by these abnormalities, which merge into the general picture of malnutrition.

The one specific clinical entity associated with protein deficiency is *nutritional œdema*, caused by the diminution in the protein of the blood plasma that results from an inadequate protein intake.

We have seen that nitrogenous equilibrium can be maintained at low levels of protein intake of the order of 40 to 50 grams, provided that the diet is of adequate Calorie value. In practice a normal diet of adequate Calorie value seldom contains insufficient protein to maintain nitrogenous equilibrium. But if the diet is deficient in Calories as well as low in protein, some of the protein is burned to produce energy and is hence not available to the body for the synthesis of body protein; so that a protein intake which was sufficient to maintain nitrogenous equilibrium on a diet of adequate Calorie value is no longer able to do so – in other words, protein deficiency is usually relative, being conditioned by an inadequate Calorie intake.

Nutritional œdema is thus an ever-present threat in any population living near the sub-nutritional borderline, and its incidence can be looked upon as an index of general nutrition, as well as of protein deficiency.

Persons suffering from nutritional œdema will therefore show, to a more or less marked degree, the evidence of general undernutrition such as emaciation (which may be masked by the œdema), atrophy of the heart and other organs, weakness, lassitude and so forth.

The essential feature of the condition is of course the œdema, which results from the fall in the colloid osmotic pressure of the plasma resulting from the diminution in the plasma proteins. The albumin fraction of the plasma proteins is affected to a much greater degree than the globulin fraction.

The œdema, being gravitational, appears first in the legs towards the end of the day, and in the face and hands in the morning. When severe, a general anasarca may develop. Although the fall in the concentration of the plasma proteins is the fundamental cause of the œdema, secondary factors such as posture, the water and salt intake, and environmental temperature (*e.g.* the increase in energy expenditure in cold weather) may determine its onset.

An often striking manifestation of nutritional œdema is the polyuria which accompanies or often precedes the appearance of the œdema. This may lead to a mistaken diagnosis of nephritis until the examination reveals a normal urine and renal function.

Protein Hydrolysates and their Use in Medicine

We have seen already that it is possible to maintain nitrogenous equilibrium in both experimental animals and men by the administration of solutions containing a mixture of the essential amino-acids. This method of feeding has an obvious application in the treatment of patients suffering from any of the numerous conditions in which there may be failure to ingest or digest protein, or to absorb the products of protein digestion.

Examples of such conditions are lesions of the throat and œsophagus, severe vomiting or diarrhœa, operations on the alimentary tract, or indeed any condition in which the patient is too ill to take ordinary food. It is now recognized that in any severe injury or operation, after the initial phase of shock, there follows a period of increased metabolic activity in which there is a marked negative nitrogen balance indicative of an extensive breakdown of body protein. In burns, the protein loss in this period of 'traumatic inflammation' is enhanced by the protein lost in the exudate. It is possible that this increased breakdown of body protein occurs primarily at the expense of the labile reserve of protein formed by those parts of the organs and tissues which can be given up without loss of function, and that this protein catabolism is necessary for the liberation of some essential amino-acids required for repair of damaged tissues.

The negative nitrogen balance is at its height about a week after the trauma has been sustained, but in serious cases it may persist for some weeks. If the patient is receiving an adequate diet, the phase of negative nitrogen balance is succeeded by one of positive balance, when the broken-down body protein is presumably regenerated.

Nevertheless, it would seem unwise to allow the reserve of body protein to be depleted, even temporarily, if it could be prevented by increasing the intake of essential amino-acids to provide material for tissue repair. Unfortunately, most patients cannot take by mouth the large amounts of protein food (often up to and even more than 150 grams daily) necessary to reduce or eliminate the negative nitrogen balance which follows injury or operation. This type of case is therefore another example of the conditions which should benefit from administration of mixtures of amino-acids.

Production of pure amino-acids is difficult and expensive, and attention has therefore been devoted to the preparation of hydrolysates of protein in which the protein has been broken down to its constituent amino-acids either by heating with acid or digestion with enzymes (pancreatic enzymes or papain). Such hydrolysates are usually made from casein; if acid is used in their preparation, tryptophane must be added to the hydrolysate since this amino-acid is destroyed during digestion with heat in the presence of acid. Protein hydrolysates suitable for both oral and intravenous administration are now available for therapeutic purposes. The intravenous method of administration has the advantage that the body cells are presented at once with a suitable mixture of amino-acids introduced into the blood stream; and it is of particular value when for any reason there is an inability, complete or partial, to ingest and digest protein or to absorb amino-acids from the alimentary tract.

Numerous experiments have shown that, provided the hydrolysate is introduced slowly into a vein, almost complete utilization of the injected amino-acids occurs. Protein feeding by the use of hydrolysates, either orally or intravenously introduced, is essentially a temporary procedure to meet certain emergencies, but patients have been maintained in nitrogen balance with hydrolysate as their only source of dietary protein for periods of several weeks.

Protein hydrolysates contain the amino-acids from only one or at most a few proteins, and therefore do not provide the mixture of amino-acids derived from dietary proteins. But the fact that nitrogen balance can be secured with enzymic casein digest (or acid digest fortified with tryptophane) indicates that this hydrolysate at least, if given in sufficient dosage (about 80 grams dry weight of hydrolysate daily), supplies the essential amino-acids in adequate amounts.

The metabolism of the amino-acids introduced into the blood stream in the form of a protein hydrolysate does not appear to differ from that of the amino-acids absorbed normally from the digestion of protein foods. For instance, the amino-acids of a protein hydrolysate are not utilized for tissue repair but are burned to produce energy if carbohydrate is not administered simultaneously. Therefore, when protein hydrolysate is given either orally or intravenously, glucose solution must be administered along with it; a 10 per cent solution is most commonly used for the intravenous route when glucose is being given along with protein hydrolysate.

A description of the use of protein hydrolysates in therapy is given in greater detail in Chapter XX.

CHAPTER IV

THE VITAMINS

With the advances in knowledge concerning the vitamins, it has become increasingly difficult to give a definition which will cover the group as a whole. They may be described as substances which the body is itself unable to synthesize from fat, carbohydrate or protein, but which are required for the maintenance of health. The brilliant researches of recent years have led to the isolation of twelve of the vitamins as pure chemical substances, and these are quite unrelated to one another chemically. Consideration of the vitamins as a group is only justified on the grounds that they occur in natural foods and the methods used for their detection and investigation are based on the same experimental principles. It is true that the vitamins can be considered as essential for life, but this generalization must be qualified when different species of animals are compared, since vitamins essential for one species may not be required as food constituents by others which are able to synthesize them from appropriate precursors. Thus, apparently the only species which cannot synthesize their own vitamin C are man, monkeys and guinea-pigs.

Views about the indispensability of vitamins in the diet have been further complicated by recent work on the synthesis of vitamins by the micro-organisms of the bowel. The poorly-absorbed sulphonamide drugs, sulphaguanidine and succinylsulphathiazole, inhibit the growth of certain of the micro-organisms which are normal inhabitants of the bowel. Symptoms of vitamin deficiency have been precipitated in animals by the addition of these sulphonamides to incomplete diets, on which, however, health had previously been maintained. By such means the synthesis of most of the vitamins of the B group and also of vitamins E and K has been demonstrated in experimental animals.

These experiments have been applied to man. Thus Najjar and Holt (1943) found that of a group of nine men fed on a diet devoid of aneurin, only five developed symptoms of aneurin deficiency, while the other four remained symptom-free. The faeces of the latter contained free aneurin in spite of the lack of this vitamin in the diet, whereas the faeces of the subjects who developed symptoms of aneurin deficiency contained no free aneurin. Administration of succinylsulphathiazole to one of the symptom-free subjects led to the disappearance of aneurin from his faeces. By similar methods, the biosynthesis of riboflavin and nicotinamide by the intestinal flora of man has been demonstrated.

A striking fact about this biosynthesis of vitamins is the great variation in the extent to which it occurs among different people even on the same diet. It is difficult to avoid the conclusion that this variation is at least part of the reason why only certain members of a group of persons eating a diet deficient in one or other of the vitamins may develop symptoms relating to deficiency of the vitamin while the other members of the group remain healthy. The phenomenon of biosynthesis of vitamins makes it easier to understand, too, why the vitamin B requirements of man have been placed at varying levels by different workers.

Although it is too early to draw final conclusions from the experiments described above, it can at least be suggested that future work may show that the actual vitamin content of a diet may sometimes be of less significance than the consumption of a diet which promotes the growth of intestinal micro-organisms with the capacity of synthesizing vitamins. In this chapter, absolute values are given for the human requirements of the various vitamins. These have been obtained by observation of diets which either prevent or precipitate vitamin deficiency, but it is obvious that some at least of the values may have to be amended considerably when more is known of the phenomenon of vitamin biosynthesis in the human bowel and the means by which this may be modified by alterations in the diet.

A further complication is introduced by the recent finding that under anærobic conditions certain types of bacteria which are normal inhabitants of the intestine possess the property of destroying vitamins, particularly nicotinic acid and ascorbic acid, and possibly vitamin B_1 also. It is possible that a balance is normally maintained between synthesis and destruction of some of the vitamins by the intestinal bacteria. Disturbance of this balance towards destruction of the vitamins by factors such as alterations in the predominant types of intestinal flora, bacterial or parasitic infections of the alimentary tract, etc., may play an important part in precipitating the development of vitamin deficiency disease. This is particularly liable to occur under conditions of stress where there is an increased requirement for vitamins.

Mention must be made here of other factors that make the establishment of precise values for the vitamin requirements difficult – namely, variations in metabolism and the presence of intercurrent disease, which by increasing the body's needs for vitamins may produce a 'conditioned' deficiency in persons consuming a diet that would be adequate under normal conditions.

The various factors predisposing to the occurrence of conditioned vitamin deficiency may be classified as follows:

(*a*) *Interference with Absorption.* For example, the presence of bile in the intestine is essential for the absorption of certain of the fat-soluble vitamins, and a patient with obstructive jaundice may develop the haemorrhagic tendency resulting from vitamin K deficiency.

(*b*) *Interference with Storage.* Certain vitamins are normally stored in the liver, but diseases of this organ may interfere with this function, and vitamin A and vitamin K deficiency may be precipitated by acute hepatitis or cirrhosis of the liver.

(*c*) *Increased Requirement.* The demands of the body for certain of the water-soluble vitamins, notably aneurin and ascorbic acid, tend to vary with the level of general metabolism, and these demands will be increased in strenuous and prolonged physical exertion, hyperthyroidism, and fever.

(*d*) *Excessive Excretion as a result of Gastro-intestinal Disease.* It is a common experience in Eastern countries that an attack of dysentery may precipitate the development of vitamin deficiency diseases, especially those relating to the vitamin B_2 complex. Consideration must therefore be given to such factors when assessing the adequacy of the diet under conditions that depart from the normal.

CLASSIFICATION

The old alphabetical order of arrangement of the vitamins is falling out of date now that several of the vitamins have received individual and chemical names. There is, however, some justification for retaining the terms water-soluble and fat-soluble vitamins, for these physical properties enable the known vitamins to be arranged into two broad categories.

The following list of the known vitamins gives both their alphabetical nomenclature, and their chemical names, where the vitamin has been isolated:

Water-Soluble Vitamins

The earliest work on accessory food factors led to the differentiation of two water-soluble vitamins known respectively as vitamin

B, the antineuritic vitamin, and vitamin C, the antiscorbutic vitamin.

It was soon found that material containing vitamin B, if subjected to heat, lost its antineuritic but retained its growth-promoting properties. This led to the sub-division of vitamin B into vitamin B_1, the heat-labile antineuritic vitamin, and vitamin B_2, the heat-stable growth-promoting vitamin. Further researches have shown that the vitamin B_2 of the earlier workers is not a single substance, but consists of probably at least eleven factors, nine of which have been obtained in crystalline form. These factors, although chemically unrelated, tend to be found in the same food materials, and are commonly grouped under the term 'vitamin B_2 complex'. There is, however, no evidence as yet that certain of these factors are essential for man.

The water-soluble vitamins can therefore be grouped as follows:

B_1 (Aneurin or Thiamin) – the antineuritic vitamin.

The B_2 Complex, consisting of:

(i) Riboflavin.
(ii) Nicotinic Acid – the pellagra-preventing factor.
(iii) Pyridoxin (Adermin, or vitamin B_6) – the rat antidermatitis factor.
(iv) Pantothenic Acid (filtrate factor) – the chick antidermatitis factor.
(v) Biotin (vitamin H) – the anti-egg-white injury factor.
(vi) Choline.
(vii) *p*-Amino-Benzoic Acid.
(viii) Folic Acid.
(ix) Inositol.
(x) Other factors of the B_2 complex – the R and S chick-growth factors.

C (Ascorbic or Cevitamic Acid) – the antiscorbutic vitamin.

P (Citrin) – a group of substances occurring in citrus fruits, and considered necessary for the stability of the capillary walls.

Fat-Soluble Vitamins

A. Carotene, known also as provitamin A, occurs in plants as a yellow pigment which is converted in the animal body to vitamin A and stored in fat. Vitamin A is necessary for the integrity of the epithelial structures.

D. Some ten closely related sterol compounds that acquire antirachitic activity when exposed to ultra-violet radiation are known, but of these only two are of importance in medicine:

1. **D_2 (Calciferol)** – a product obtained by the ultra-violet irradiation of ergosterol.
2. **D_3** – an antirachitic vitamin found in fish-liver oils, and also prepared artificially by the irradiation of 7-dehydrocholesterol.

E. Tocopherol. The anti-sterility factor.

K. Required to maintain normal blood-clotting powers in growing chicks.

In the following more detailed consideration of the vitamins, stress will be laid only on those whose importance in human nutrition has been established.

THE WATER-SOLUBLE VITAMINS

Vitamin B_1 (Aneurin or Thiamin)

Chemical and Physical Properties. Vitamin B_1 in the form of aneurin chloride hydrochloride* is a slightly acid, white crystalline substance. It is readily soluble in water, but is rapidly destroyed by heat in neutral or alkaline solutions; in acid solution, however, it is resistant to heat up to 120° C.

Physiological Function. In the body tissues, aneurin occurs both in the free form and as a compound, aneurin pyrophosphate. In the latter form it functions as a co-enzyme (cocarboxylase) which in combination with an enzyme system brings about the breakdown of pyruvic acid, one of the intermediate stages in the oxidation of glucose. In aneurin deficiency, the metabolism of carbohydrate is interfered with, and pyruvic acid accumulates in the tissues and body fluids. This increase of pyruvic acid can be detected in the blood in human cases of beri-beri and is sometimes employed as a method of diagnosing aneurin deficiency, although the use of this

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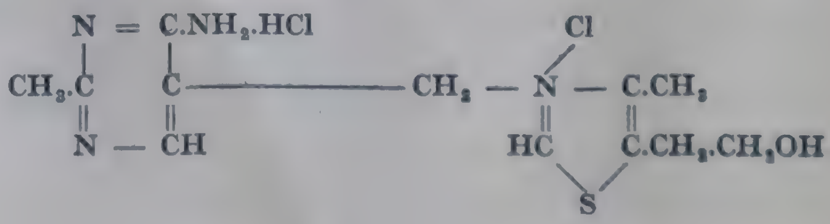
* Aneurin chloride hydrochloride is built up of two ring systems, a pyrimidine ring and a thiazole ring. It has the following structural formula :



test has been criticized on the ground that the resting level of pyruvic acid in the blood is raised only in severe degrees of aneurin deficiency. At one time it was thought that the symptoms of aneurin deficiency might be due to the toxic action of this accumulation of pyruvic acid in the tissues, but the modern view is that the failure of normal carbohydrate metabolism, and hence of energy supply to the tissue cells, that occurs in aneurin deficiency is in itself sufficient to account for the dysfunction found in this condition.

Symptoms of Deficiency. Although the function of aneurin in the body has been elucidated, little is known of the mechanism whereby a deficiency produces the characteristic changes that occur both in man and in experimental animals. All that can be said is that aneurin appears to be essential for the health of nerve tissue and for normal cardiac and gastro-intestinal function.

The most constant lesions in aneurin deficiency in man are found in the peripheral nerves and the heart, although none of these is specific. This predilection may possibly be attributable to the fact that only carbohydrate is burned in the energy metabolism of nerve tissue so that there must be a special need for cocarboxylase there, while the constant muscular activity of the heart may well give rise to another special need for the co-enzyme. The peripheral nerves first show a degeneration of the myelin sheath, followed by fragmentation of the axis cylinders and Wallerian degeneration. The nerves of the legs are usually affected first and the degenerative process begins in the distal portion of the nerve; muscles supplied by the affected nerves undergo the usual atrophy found in other forms of neuritis. The most prominent pathological change in the heart is a great enlargement, especially of the right side, due to dilatation and increased thickness of the wall of the right ventricle and distension of the right auricle. Associated with the cardiac enlargement there is usually œdema, both subcutaneous and involving the body cavities, most probably due to congestive cardiac failure, although some workers consider it to be due to a specific effect of aneurin on the capillaries.

In experimental animals suffering from aneurin deficiency, functional and organic lesions of the gastro-intestinal system have been described – reduced gastric acidity, gastro-intestinal atony, degeneration of Auerbach's plexus and, according to some workers, gastric erosions and ulcers. Many of these studies have been complicated by the inadequacy of factors other than aneurin, and the precise relationship of aneurin to gastro-intestinal function still awaits clarification. There is, however, convincing evidence that aneurin deficiency is characterized by marked anorexia.

In its fully developed form, beri-beri, the name given to the group of symptoms found in severe aneurin deficiency, is almost confined to those parts of the East where polished rice forms the staple article of diet. Under ordinary conditions of Western civilization, frank beri-beri is seen only sporadically in persons whose diet is deficient in aneurin through food faddism, mental disorder, alcoholism, etc., but minor degrees of aneurin deficiency were probably not uncommon in this country before 1942, when the extraction rate of flour was increased as a wartime measure.

While the difference is one of degree only, there is some justification for describing the less striking manifestations of mild aneurin deficiency separately from the symptoms of classical beri-beri, in that the former are much more likely to be encountered by the physician.

(*a*) *Mild Aneurin Deficiency.* The symptoms are non-specific and not striking, but their occurrence in circumstances likely to lead to a deficient aneurin intake and where they cannot otherwise be explained should lead the physician to consider the possibility of aneurin deficiency. Such circumstances include factors leading to a defective dietary intake – ignorance, poverty, or disease affecting the appetite, intake and absorption of food ; or factors increasing the aneurin requirement, such as pregnancy and fever.

The clinical manifestations of mild aneurin deficiency can be divided into four groups:

(i) *General.* Weakness, easy fatiguability, irritability and nervousness, headaches, insomnia and loss of weight. It should be noted that these clinical features can equally well be ascribed to a psychoneurosis.

(ii) *Neurological.* Stiffness and cramps of the legs, sometimes amounting to actual neuritic pain, and paræsthesiæ of the legs and feet. Physical examination may reveal tenderness of the calf muscles on pressure, areas of hyperæsthesia and diminished vibratory sensibility in the legs and diminution or loss of the patellar and ankle tendon reflexes. Weakness of the legs is often brought out by the so-called 'squatting test' where the patient is asked to rise from a squatting position.

(iii) *Cardio-vascular.* Palpitation, dyspnœa, and sometimes slight œdema of the legs. Signs are usually absent until the degree of aneurin deficiency is bordering on frank beri-beri, of which the earliest cardio-vascular signs are slight enlargement of the heart, especially to the right, undue tachycardia on exertion, variable systolic murmurs, exaggerated venous pulsations, with sometimes a slight elevation of the systolic blood pressure.

(iv) *Gastro-intestinal.* Anorexia, indigestion and constipation.

Two types of aneurin deficiency formerly classed as separate forms of neuritis merit special attention – alcoholic polyneuritis and the polyneuritis of pregnancy.

Alcoholic neuritis is now accepted as being due to aneurin deficiency, and not to any direct toxic action of alcohol, since it can be cured by administration of aneurin even when patients are allowed to continue their usual high intake of alcohol. The intake of aneurin in chronic alcoholism may be low, since alcoholic polyneuritis is especially common among elderly males living alone and cooking their own food. It may, however, be within the accepted normal limits and yet be relatively deficient, being sufficient to cover the Calories consumed as food, but not the additional Calories ingested in the form of alcohol. The gastro-intestinal disorders of the chronic alcoholic may be an additional factor causing interference with the absorption of aneurin, and so contribute to the development of a conditioned aneurin deficiency.

Polyneuritis of Pregnancy. As in the case of alcoholic polyneuritis this condition was formerly ascribed to a toxin, but it has now been clearly shown to be due to aneurin deficiency. Polyneuritis in pregnancy is often associated with vomiting, especially the pernicious vomiting of pregnancy, but it is not yet certain to what extent the aneurin deficiency giving rise to pregnancy polyneuritis is due primarily to the vomiting, or to such additional factors as anorexia, gastro-intestinal dysfunction and the great demands made by the fœtus on the mother for aneurin. Probably all factors are operative.

(*b*) *Beri-beri.* Severe aneurin deficiency in its chronic form has a gradual onset with a prodromal period marked by the vague symptoms and signs of mild aneurin deficiency described above. Later, major symptoms and signs appear, which have been used to divide classical beri-beri into its two primary forms, dry or wet beri-beri.

(i) *Dry beri-beri* is marked by the symptoms of a neuritis, beginning in the legs but later extending upwards to involve the upper extremities and even the trunk muscles, with the usual features of weakness, muscle tenderness, paræsthesiæ, ataxia due to loss of sensory impulses, diminution or loss of tendon reflexes, and atrophy of muscles, leading to foot drop and contracture deformities. Recovery from anything but a mild degree of this condition is slow, and in severe cases muscle atrophy and weakness may persist indefinitely.

(ii) *Wet beri-beri.* The most prominent feature is œdema, beginning in the legs, but often progressing to generalized anasarca. Accompanying the œdema are other symptoms of congestive heart failure, and the heart is greatly enlarged, especially to the right.

Wet beri-beri may be accompanied by the typical peripheral neuritis of the dry form, giving a mixed type of the disease. While the onset is usually gradual as in the dry form, wet beri-beri may develop acutely with the rapid appearance of gross œdema. Sudden cardio-vascular collapse and death may occur even in mild cases of wet beri-beri and sometimes in patients with only neuritic manifestations.

In addition to the peripheral neuritis and cardio-vascular symptoms beri-beri is accompanied by severe anorexia, and sometimes by nausea, vomiting and constipation, especially in the acute cases. Gastric acidity is reduced.

Requirements and Dosage. Before aneurin had been isolated and synthesized in pure form, its dosage was expressed in terms of International Units, based on the potency of a standard concentrate obtained by adsorption of the vitamin on fuller's earth. Now that aneurin can be prepared in crystalline form, the International Unit of vitamin B_1 is taken as 3/1000 milligram (3 micrograms) of aneurin hydrochloride, so that 1 milligram of aneurin hydrochloride equals 330 International Units of vitamin B_1. The requirements and dosage of vitamin B_1 are now commonly expressed in terms of milligrams of aneurin hydrochloride.

It is generally accepted that in animals the aneurin requirement is related to the Calorie intake and perhaps also to the body weight. It has long been known that the needs of animals are higher on a diet containing a large proportion of carbohydrate than on a diet high in fat, and this has been attributed to an aneurin-sparing action of fat. Efforts to prove that the aneurin requirement of man varies with the proportion of carbohydrate and fat in the diet have not given uniform results, and man's needs for aneurin are therefore still commonly assessed on the total Calorie intake. The minimum requirements of an adult are usually given as about 10 International Units (0·03 mg.) per 100 Calories, a figure which is supported by several reports of the development of beri-beri in groups of men whose aneurin intake did not reach this figure. The optimum requirements (*i.e.* the level above which no further increase in the vitamin produces any improvement in health) have been placed at 15 to 20 International Units (0·045 to 0·06 mg.) per 100 Calories, *i.e.* about 450 to 600 International Units (1·4 to 1·9 mg.) for an average man. Children and pregnant and lactating women appear to need more aneurin in relation to their Calorie intake than adults, and for these special classes the optimum requirements have been placed at 25 to 33 International Units (0·08 to 0·1 mg.) per 100 Calories, *i.e.* about 600 to 800 International Units (1·9 to 2·5 mg.) for a pregnant woman.

In the treatment of established aneurin deficiency, much larger amounts are required. Doses of 10 to 20 milligrams (3,300 to 6,600 International Units) of crystalline aneurin for cases of beri-beri of average severity, or even up to 40 milligrams (approximately 13,000 International Units) in severe cases, should be given daily, by mouth or parenterally. The latter route should be used if interference with ingestion or absorption is present as a result of vomiting or diarrhœa. After the major symptoms have subsided, the crystalline vitamin may be replaced in whole or in part by concentrates rich in aneurin (*e.g.* yeast, wheat germ, or rice polishings) in amounts sufficient to yield a total daily intake of about 5 to 10 milligrams (1,650 to 3,300 International Units) of aneurin. Vitamin therapy should be supported by a generous diet, rich in other vitamins besides aneurin, since most patients with aneurin deficiency suffer from other nutritional disorders as well. In severe cases with marked neuritis, treatment may have to be continued for months before complete recovery is effected.

Natural Sources. Aneurin is widely distributed in nature, but no foods commonly eaten are very rich in aneurin, and the daily supply must therefore be secured from a good mixed diet of natural foodstuffs. It is when processed foods like white bread, polished rice and sugar form a large part of the diet that aneurin deficiency is likely to occur.

The two richest natural sources of aneurin are yeast and the germ of cereals, or rather a particular portion of the germ. Common foods containing relatively large amounts of aneurin are whole-grain cereals, eggs, legume vegetables, nuts and fresh meat, especially the viscera. Of the muscle meat, pork is a much richer source of aneurin than beef or mutton. Milling of cereals below an extraction rate of about 75 per cent removes the germ, so that white flour and polished rice retain little of the aneurin of the whole-grain cereal.

As pointed out in Chapter XXI, the intake of any nutrient in a particular foodstuff depends not only on its concentration in the foodstuff but on the amount of this foodstuff in the diet. From this point of view, potatoes and vegetables, although relatively poor in aneurin, contribute a significant amount towards the daily intake. The great importance of bread baked from high-extraction flour as a source of vitamin B_1 is discussed in detail in Chapter VIII.

Aneurin is readily soluble in water, and considerable amounts are therefore extracted when foodstuffs are cooked in water; this is especially applicable to the vegetables. Aneurin is, however, relatively stable at temperatures up to boiling point except in cooking under pressure, so that there is little actual destruction, and the aneurin extracted into the cooking water will be saved if this is used as stock. Aneurin is rapidly destroyed in alkaline solution, and for this reason the use of soda in the cooking of green vegetables is very detrimental. Similarly baking with baking powder, which produces an alkaline dough, causes considerable destruction of the aneurin in the flour; on the other hand, baking with yeast, wherewith the dough is acid, is much less destructive. It is wise to reckon that the loss of aneurin from the cooked foodstuffs of an ordinary mixed diet amounts to about 25 per cent on the average.

Proprietary Preparations. Where vigorous aneurin therapy is indicated, preparations of three kinds are available; these either contain crystalline aneurin, or consist of concentrates made from naturally rich sources of aneurin, or are composed of yeast.

(*a*) *Preparations containing Crystalline Aneurin.* Among the many products of this nature may be mentioned:

(i) For oral administration:

Benerva (Roche) Berin (Glaxo) Betalin (Lilly) Tabloid Vitamin B_1 Hydrochloride (Burroughs Wellcome) Vitamin B_1 (British Drug Houses)	All contain 1 mg. aneurin per tablet.

(ii) For parenteral administration:

Benerva (Roche) – ampoules containing 2 mg. or 10 mg. aneurin.
Berin (Glaxo) – ampoules containing 2 mg. or 10 mg. aneurin (Berin Forte.)
Betalin (Lilly) – ampoules containing 1 mg., 6 mg., 10 mg., or 30 mg. aneurin.
Betaxan (Bayer Products) – ampoules containing 2 mg. or 10 mg. aneurin.

(*b*) *Preparations made from Naturally Rich Sources of Aneurin.* A number of such specially prepared products are available for treatment that falls between the prophylactic and the therapeutic. Among these the best known are 'Bemax', a preparation made from wheat germ, which contains 300 to 400 International Units (approximately 1·0 to 1·2 mg.) of aneurin per ounce, and 'Marmite', a yeast extract containing about 150 International Units (approximately 0·5 mg.) of aneurin per ounce. The latter cannot, however, be taken by most patients in amounts greater than two or three teaspoonfuls daily, and the importance of 'Marmite' as a dietary supplement therefore lies more in its high content of factors of the vitamin B_2 complex.

(*c*) *Yeast.* Although yeast, in strict nutritional parlance, can be classed as a foodstuff, it can hardly be regarded as such in the ordinarily accepted sense of the term. In practical dietetics, it should be looked upon as a food supplement whose value lies in its high content of aneurin and the other vitamins of the B complex and to a somewhat lesser extent in its richness in protein of high biological value, the protein content of yeast being about 40 to 50 per cent of the dry matter. The amount of aneurin in yeast depends to a large extent on the nature of the medium in which the yeast is grown, but the amounts of riboflavin and nicotinic acid, and probably also of the other members of the vitamin B_2 complex, do not vary so much among the different types of yeast. The nutritive value of these is given in detail in the Table of Food Values (see page 435). Two forms of yeast require consideration in dietetics – ordinary baker's or brewer's yeast (saccharomyces cerevisiæ) and the species known as 'food yeast' (torulopsis utilis), the commercial production of which is being initiated on a large scale.

(i) *Baker's or brewer's yeast.* This is obtainable either as fresh or as compressed yeast, or in dried form.

Brewer's yeast contains more aneurin than baker's yeast. A baker's yeast specially rich in aneurin has, however, been developed by growing it on a medium to which the chemical components of aneurin have been added, but this naturally increases the cost of the product.

Brewer's yeast, and to a less extent baker's yeast, has a bitter salty taste, which limits the amount that can be ingested. Compressed yeast has a high water content (70 per cent in baker's yeast, 75 to 85 per cent in brewer's yeast) and does not provide much more aneurin than can be obtained from the same weight of bread made from high-extraction flour. There is evidence, too, that the absorption and utilization of aneurin from fresh (*i.e.* compressed) yeast is less complete than from dried yeast; indeed, very little aneurin may be absorbed from fresh yeast. It must therefore be looked upon chiefly as a source of vitamins of the B_2 complex, and even so, relatively large amounts – 2 oz. upwards daily – must be prescribed if therapeutically significant amounts of these vitamins are to be administered. It is best given in divided doses of not more than 1 oz. mixed in a tumblerful of fruit juice (*e.g.* prune juice) or tomato juice to form a suspension, by which means its bitter taste is somewhat disguised. The easiest way to incorporate compressed yeast in fruit juices is to adopt the procedure followed in bread making. The amount of yeast to be used should be placed in a small basin with $\frac{1}{8}$ of the quantity of sugar and the two mixed together until the yeast becomes liquid, when it can be added to the fruit drink in which it is to be given.

Fresh yeast is very apt to cause digestive upset, and for this reason dried yeast is preferable as a therapeutic agent. Boiling suspensions of fresh yeast before incorporating them in the fruit or tomato juice vehicle, so as to kill the yeast cells, will reduce the tendency to digestive upset, and will also increase the amount of aneurin absorbed from the yeast.

Dried yeast contains only 5 to 10 per cent of water, but amounts of 1 oz. and upwards daily are necessary to secure an intake of B vitamins in therapeutically significant amounts, except in the case of the special aneurin-rich yeast mentioned above, which contains about 300 International Units of aneurin per gram. Dried yeast can be obtained either in powdered form or in tablets. The latter contain only a few grains of dried yeast, however, so that with the exception of the special aneurin-rich yeast, the use of yeast tablets in the treatment as distinct from the prophylaxis of vitamin B deficiency diseases is not practicable.

Powdered dried yeast should be given in fruit or tomato juice, in sweetened milk, or in water flavoured with tomato catsup, to mitigate somewhat the unpleasant, bitter taste of the yeast. Individual doses should not exceed a tablespoonful in a tumblerful of vehicle, and the yeast should be sifted into the fluid in which it is to be given, while stirring constantly with a spoon or preferably a rotary egg-beater.

It must be admitted that most patients find yeast unpleasant to the taste, and that it is difficult to persuade them to take it. Also, even dried yeast, if given in amounts above about ½ oz. daily, tends to cause digestive upset.

(ii) *Food Yeast.* Unlike baker's or brewer's yeasts, which are essentially by-products of industrial processes, food yeast is being developed deliberately for the nutritional purpose of providing a food supplement rich in vitamins of the B complex and good quality protein. It is cultivated in a medium of raw cane sugar, molasses, or sugar juice, to which ammonium sulphate and superphosphates are added.

Apart from commercial advantages such as ease and economy of manufacture, food yeast has one great advantage dietetically over other types of yeast – it lacks the bitter taste of the other varieties, and being more palatable as a result, can be consumed in larger quantities.

Food yeast is being made available only in the dried form as a powder, which can be introduced into all kinds of savoury preparations like soups, stews, gravies and sauces, adding richness to the flavour – a tablespoonful (½ oz.) of dried food yeast per pint of liquid is the strength recommended. Food yeast can also be added, at concentrations up to 5 per cent, to the flour used for baking bread or biscuits, without detracting from the flavour or quality of the product, or it may be sprinkled as a garnish on vegetable salads, stewed fruit, etc. It can of course be given as a suspension in fruit juice, as was recommended for other forms of yeast. One ounce of food yeast will supply almost 200 International Units of aneurin, about 1·4 milligrams of riboflavin and almost 12 milligrams of nicotinic acid, so that 1½ oz. daily would alone provide the minimum daily requirement of aneurin, and the optimal requirements of riboflavin and nicotinic acid. Food yeast, owing to its palatability, is therefore to be regarded as the yeast preparation of choice for nutritional purposes, and it is hoped that it will not be long before it is universally available for the prophylactic and therapeutic administration of the B vitamins in natural form.

B_2 Complex

As we have seen, what is known as the vitamin B_2 complex is really a group of at least eleven chemically unrelated substances, whose association under a single term is due to the fact that they tend to occur in the same foods; their biological activity was at first ascribed to a single substance, vitamin B_2, until progressive biochemical and biological researches identified the different vitamins and their actions in the body. The latest investigations of the functions of the various substances grouped under the term 'vitamin B_2 complex' suggest, however, that there is some justification for this generic nomenclature. Several of the enzymes concerned in the oxidation-reduction reactions of living cells are conjugated proteins consisting of a prosthetic group containing one of the vitamins of the B_2 complex attached to a protein carrier; these either exert enzymatic activity themselves or are necessary for the activation of enzymes. Also, all the members of the vitamin B_2 complex appear to have one function in common, that of stimulating metabolism and thereby promoting growth, either of higher animals or of micro-organisms.

Of the different members of the B_2 complex, only two – riboflavin and nicotinic acid – have definitely been proved to be essential in human nutrition.

Riboflavin (*Lactoflavin*)

Chemical Nature and Properties. Riboflavin * is a yellow pigment occurring in plants and animals; it displays a characteristic yellowish-green fluorescence in aqueous solution. It has been known for a long time as a constituent of plant and animal foods, but only within recent years has its importance as a vitamin been recognized. It is soluble in water, and is resistant to heat especially in an acid medium, but is readily decomposed by visible and ultra-violet light.

Physiological Functions. In natural sources, riboflavin either occurs in the free form, or combined with phosphoric acid, as an ester (riboflavin phosphate), which alone or in combination with similar substances unites with certain proteins to form a group of enzymes concerned with the oxidation-reduction reactions within the cell that lead to the breakdown of food substances such as glucose.

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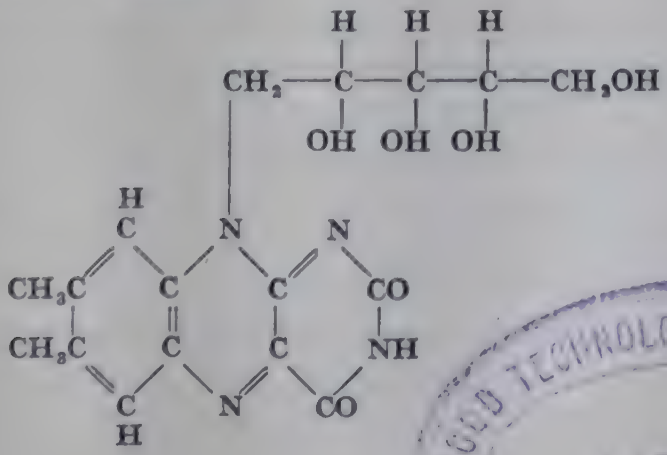
* Riboflavin consists of a tricyclic nucleus, dimethyl isoalloxazine, to which is attached a side-chain composed of a molecule of ribitol (the alcohol derived from the pentose sugar, ribose). It has the following structural formula :



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The first of these enzymes to be discovered was the 'yellow oxidation enzyme' of Warburg, which is present in varying amounts in practically all living cells and plays a significant part in cell respiration; other flavo-proteins like xanthine oxidase have similar functions.

The presence of riboflavin in every living cell, at least in the higher forms of life, suggests that it must be essential to man and other animal species. Little is known about the absorption, storage and excretion of riboflavin in man, but on a normal diet amounts in the neighbourhood of 1 milligram daily are excreted in the urine, and balance experiments have indicated that a certain storage of riboflavin can occur in the body, although there is no evidence that such storage represents a functional bodily requirement.

Symptoms of Deficiency. Although the relation of riboflavin to cellular respiration has been elucidated, little or nothing is known of the manner in which a deficiency of the vitamin produces the pathological changes which can be recognized clinically.

In animals, deprivation of riboflavin causes failure of growth, but specific lesions vary in different species. In the rat, eye lesions, including corneal opacity and vascularization, and denudation of the hair and a moist eczema with scaliness, are the most prominent features of riboflavin deficiency, whereas in other species (dog, pig, and chicken) nerve degeneration and paralysis predominate.

Within recent years, riboflavin deficiency in man has been shown to give rise to a definite syndrome characterized by lesions of the lips, tongue, and skin, and also of the eyes, although the causal relationship of riboflavin deficiency to the ocular lesions is a matter of controversy.

(*a*) *Mouth Changes*

Burning and soreness of the lips and tongue, and soreness at the corners of the mouth accompany the lesions of the mouth and tongue. The characteristic lesions are:

(i) Cheilosis. The buccal aspects of the lips become unduly reddened, superficial denudation of the epithelium occurs, and in severe cases there may be shallow ulceration and crusting.

(ii) Angular stomatitis. At the angles of the mouth the epithelium becomes whitened, thickened and sodden looking. Shallow transverse fissures appear, radiating from the corners of the mouth; these are often covered with yellowish crusts and leave shallow, linear scars when they heal. The whitened, sodden epithelium often extends backwards from the corners of the mouth as a linear lesion along the prolongation of the vermilion portion of the lips on to the buccal surface of the cheeks.

(iii) Glossitis. The characteristic tongue lesion in riboflavin deficiency is said to be a purplish-red discoloration of the tongue which presents a clean, but deeply fissured surface with enlarged, flattened, mushroom-shaped papillæ. The filiform papillæ lose their processes and later atrophy, leaving a smooth surface, often with a patchy distribution – the so-called 'geographical tongue'.

(*b*) *Skin Changes*

The skin of the naso-labial and naso-malar folds, the alæ nasi, the vestibule of the nose, and the ears may show a fine, scaly seborrhœic dermatitis, which is sometimes accompanied by a peculiar follicular hyperkeratosis over the nose and naso-labial folds, with pointed horny plugs projecting from the enlarged pilosebaceous follicles.

(*c*) *Ocular Lesions*

Symptoms and signs referable to the eyes have been described by several writers as being associated with cheilosis, glossitis, and other symptoms of riboflavin deficiency. The symptoms include photophobia, burning and itching of the eyes, poor vision in a dim light, and conjunctivitis, while the characteristic sign is stated to be corneal vascularization. The process begins with a congestion of the limbic capillary plexus lying at the sclero-corneal junction, this being followed by a centripetal invasion of the superficial layers of the cornea by small capillaries arising from the loops of the limbic plexus all round the circumference of the cornea. As the lesion progresses, the invading capillaries form anastomotic loops from which further capillary twigs pass further inwards. Following this corneal vascularization comes opacity of the cornea due to superficial nebulæ which give the surface of the cornea a steamy look. These lesions have been stated by many workers to be diagnostic of riboflavin deficiency and curable by large doses of riboflavin, some observers claiming that symptoms disappear and the capillaries become emptied of blood within a few days whereas others state that treatment may have to be prolonged for anything up to two months.

The occurrence of varying degrees of corneal vascularization in persons whose dietary records indicated that they had been receiving an adequate supply of riboflavin, the failure of dietary surveys to show any clear correlation between riboflavin deficiency and corneal vascularization, and the resistance shown by corneal vascularization unaccompanied by other signs of riboflavin deficiency to specific vitamin therapy, have combined to cast doubt on the specificity of the corneal lesion as a diagnostic sign of riboflavin deficiency. Lyle and his co-workers (1944), who examined groups of men on varying diets and under different climatic conditions, found that the incidence of corneal vascularization was highest where the diet was poor in fresh vegetables and fruits, and that improvement in the condition was much more marked on a diet rich in yeast, milk, fruit juices, and vegetables than with the administration of pure riboflavin, suggesting that some nutritional factor other than riboflavin may be involved in the production of corneal vascularization.

In the present state of knowledge, it is probably safest to conclude that while corneal vascularization may occur in riboflavin deficiency and be cured by treatment with the vitamin, the condition cannot always be attributed, at least directly, to riboflavin deficiency, and cannot therefore be considered as pathognomonic of this deficiency disease.

Workers in many parts of the world have described syndromes in which the cheilosis and angular stomatitis now known to be characteristic of riboflavin deficiency have been associated with other symptoms – an eczematous lesion of the scrotum, and a group of neurological symptoms including retro-bulbar neuritis, deafness, ataxia, paræsthesiæ, and a sense of muscular weakness. Stannus (1944) has claimed that there are grounds for believing that all these signs and symptoms are manifestations of riboflavin deficiency, and are explicable on the basis of his hypothesis that the fundamental lesion in riboflavin deficiency is a disturbance in the function of the endothelial lining of the capillaries. This leads to capillary dilatation and decreased blood flow, and secondarily to metabolic disorder in the cells of the neighbouring tissues. But the possibility cannot be excluded that some of the symptoms in this syndrome may be due to deficiency of other members of the vitamin B_2 complex – one worker indeed has stated that the symptoms are improved by riboflavin administration but cured by yeast extract.

Lesions identical with those of riboflavin deficiency were described many years ago as occurring in association with the more characteristic symptoms of pellagra. It is probable that these are signs of a riboflavin deficiency complicating pellagra, since the type of diet eaten by pellagrins is likely to be deficient in more than one nutrient factor.

Riboflavin deficiency may sometimes be present as a conditioned deficiency. There seems little doubt, for instance, that the angular stomatitis occurring in sprue is an example of this.

Requirements and Dosage. From evidence gained in studies on the excretion of riboflavin in human subjects, the first authoritative pronouncement, that made by the Food and Nutrition Board of the United States National Research Council, defined the daily requirements of riboflavin as lying between 2·2 and 3·3 milligrams for an adult man, depending on the Calorie intake, with lesser amounts for women and children. But in earlier experiments on the inducement of riboflavin deficiency in man, in which symptoms of cheilosis and angular stomatitis developed in about four months on a diet yielding somewhat less than 0·5 milligrams riboflavin daily, cure of the symptoms of deficiency was effected in a few days with doses of only 1 to 2 milligrams riboflavin daily. The maintenance intake of a nutrient is almost always lower than the curative dose, and on this premise, the daily requirement would be expected to be somewhat less than 2 milligrams daily. In fact, Keys and his co-workers (1944) were unable to find any clinical or biochemical abnormality in men who had been kept on an intake of approximately 1 milligram riboflavin daily for five months, apart from a reduction in the urinary excretion of riboflavin during the first few weeks to a minimum level which thereafter remained approximately constant throughout the experimental period. It is of course not safe to argue that such a low intake can be continued indefinitely without harm, and other workers have claimed that an intake of at least 1·5 milligrams of riboflavin daily is necessary to prevent progressive depletion of the body stores.

The available evidence therefore suggests that an adequate intake of riboflavin for the maintenance of health is provided by 1·5 to 2·0 milligrams per day. As in the case of the other vitamins, the needs of the priority classes – children and pregnant and lactating women – may be greater.

Curative treatment demands bigger doses, although in mild cases doses of 3 to 5 milligrams daily are usually sufficient. In severe cases, doses of up to 30 milligrams daily may be required, and it may be necessary to give riboflavin parenterally where difficulty in absorption from the gastro-intestinal tract is suspected.

Natural Sources. Riboflavin is widely distributed among plant and animal foods, but in most it is present in only small amounts. The richest sources, apart from yeast, are milk, cheese, eggs and meat offals, while muscle meat, fish, wholemeal cereals and the pulses are less rich, but still good sources. Ale and beer contain not insignificant amounts, and a pint of strong ale would provide almost a quarter of the daily requirement of the vitamin.

In a well-balanced Western European diet, dairy products supply up to half, meat products about a quarter, and bread and cereal products, if made from high-extraction flour, about one-sixth of the total daily riboflavin intake.

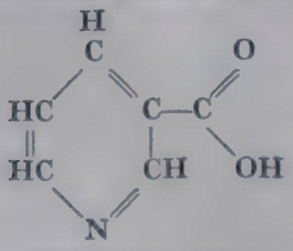
Proprietary Preparations

Beflavit (Roche)	1mg. and 3 mg. tablets. 10 mg. ampoules.
Riboflavine (B.D.H.)	1 mg. tablets. 1 mg. ampoules.
Riboflavine (Burroughs Wellcome)	'Tabloid', 1 mg. and 5 mg. per tablet. 'Hypoloid' ampoules containing 1 mg.

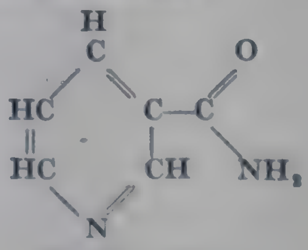
Nicotinic Acid (Niacin)

Chemical Nature. Nicotinic acid * is a simple derivative of pyridine. Although related chemically to nicotine, it possesses very different physiological properties, and is non-toxic even in large doses. Pure nicotinic acid is a white crystalline substance

* Nicotinic acid is beta-pyridine carboxylic acid. The structural formulae of the acid and of the amide, the form in which it occurs in co-enzymes I and II in the body, are as follows :



Nicotinic Acid.



Nicotinamide.

readily soluble in water; both in the dry state and in solution it is resistant to heat.

Physiological Function. Interest in nicotinic acid was aroused many years ago when it was found to be a component of extracts of rice polishings or yeast, and an unsuccessful attempt was made to identify it with the anti-neuritic vitamin. Interest revived when it was shown in 1935 that nicotinic acid was a constituent of one of the co-enzymes concerned in the oxidation of carbohydrate. Two years later it was found that black-tongue, a disease of dogs common in localities where pellagra was endemic and pathologically similar to this disease, was curable by nicotinic acid, and the evidence of its effectiveness in pellagra was soon forthcoming.

Nicotinic acid in the form of the amide is combined in the body with phosphoric acid, a purine base (adenine), and a sugar (ribose), to form two of the co-enzymes participating in the intermediary metabolism of carbohydrate.* Lack of nicotinic acid in the diet prevents the formation of these co-enzymes, since the body cannot synthesize nicotinic acid. The mechanism whereby a disturbance of this enzyme system leads to the degenerative changes in epithelial and nervous tissues characteristic of pellagra is unknown.

Symptoms of Deficiency. We have seen that the classical symptoms of the human disease, pellagra, are amenable to treatment with nicotinic acid given in adequate doses. Pellagra is therefore almost certainly due to a deficiency of nicotinic acid in the diet, although earlier theories postulating a toxic or an infective cause are still sometimes advanced.

Pellagra occurs in endemic form chiefly in maize-eating communities, especially in the countries around the Mediterranean and Black Seas, and in the southern part of the United States. It is estimated that until recently as many as 200,000 cases have occurred annually in the latter country. The consumption of maize is not essential for the development of pellagra, however, and it has been observed in rice-eating native communities, polished rice, like maize, having a low nicotinic acid content.

* Co-enzyme I (diphosphopyridine nucleotide or cozymase), containing two molecules of phosphoric acid, is a hydrogen carrier in sugar fermentation, muscle contraction, and tissue oxidation, and Co-enzyme II (triphosphopyridine nucleotide), containing three molecules of phosphoric acid, is a hydrogen carrier in the dehydrogenation of glucose, hexose monophosphate, and other substances. The most reactive part of the molecule of either co-enzyme is the nicotinamide component, which acts as the hydrogen carrier, reversibly taking on hydrogen.

The characteristic pathological changes in pellagra are an inflammatory lesion of the skin, most marked on parts of the body exposed to light, inflammation of the mucous membrane of the alimentary tract from the mouth to the rectum, and lesions of the central nervous system consisting of scattered degeneration of axis cylinders in the pyramidal tracts and myelin degeneration of fibres of the posterior columns of the spinal cord. A porphyrinuria often occurs in pellagra and it has been suggested that this excess of blood porphyrin is responsible for the photosensitivity of the skin. Porphyrinuria is, however, an inconstant finding.

The characteristic clinical manifestations are usually preceded by a prodromal period of indefinite symptoms of weakness and lassitude, mental depression or irritability, anorexia, dyspepsia and a tendency to diarrhœa. In the fully developed disease, the symptoms relate to the digestive system, the skin and the nervous system.

1. *Digestive System*

Glossitis and Stomatitis. A sore tongue is one of the first signs of the disease, and the mouth symptoms vary with the severity of the disease from a mild redness, soreness and smoothness of the tongue, first evident at the tip and along the sides, to a very severe inflammation with fiery redness of the tongue, atrophy of the lingual papillæ leaving a smooth glazed surface, and dryness, redness and ulceration of the buccal mucosa.

Diarrhœa. This symptom varies in severity from several loose stools a day to a profuse watery diarrhœa, sometimes with blood and mucus in the stools and accompanied by tenesmus. There is often severe proctitis, with redness and itching and burning sensations about the rectum.

Nausea and vomiting are common in the severe cases.

2. *Skin*

The earliest sign is an erythema resembling sunburn, occurring characteristically with a bilateral symmetry over the exposed parts of the body, especially the backs of the hands, the wrists and forearms and in the region of the sternal notch.

The skin in the affected areas is at first red and slightly swollen, with a sharply defined, slightly raised border; it itches and burns. In acute cases, the skin lesion may progress to vesiculation, cracking, exudation and crusting, but in chronic cases or in the healing of acute cases, the dermatitis occurs as a roughening and thickening of the skin with dryness, scaling and a brown pigmentation.

3. *Nervous System*

In the milder cases, the symptoms consist chiefly of psychoneurotic manifestations such as depression, irritability and failure of concentration and memory; in more severe cases, delirium is the commonest mental disturbance in the acute form of the disease, and dementia in the chronic form.

Acute cases often show paræsthesiæ and loss of tendon reflexes, but these are probably due to an associated aneurin deficiency. In chronic cases, symptoms of posterolateral degeneration of the spinal cord (spastic paresis, ataxia and loss of sphincter control) occur, being most likely manifestations of the lesions of the central nervous system caused by deficiency of nicotinic acid itself.

Anæmia, usually macrocytic, is present in about half the cases of pellagra. This anæmia, is, however, most probably not a manifestation of nicotinic acid deficiency in itself, but due rather to associated deficiencies, possibly including the anti-pernicious anæmia factor, since it responds to the parenteral injection of purified liver extract.

Lesions such as cheilosis, angular stomatitis, and scrotal dermatitis, frequently described as occurring in pellagra along with the more characteristic symptoms mentioned above, are now considered to be most probably due to a complicating riboflavin deficiency.

Requirements and Dosage. It has been found difficult to establish precise figures for the nicotinic acid requirements in man – balance experiments, for instance, being complicated by the fact that excess nicotinic acid is excreted as derivative substances as well as the acid itself. As shown in Table I (page 2), the average requirements of an adult have been placed at 15 to 20 milligrams, and the United States Food and Drug Administration has set the minimum daily requirement at 10 milligrams.

Much larger doses are required in the treatment of established cases of pellagra, and 200 to 500 milligrams of nicotinic acid daily in divided doses are recommended for moderate and severe cases. Nicotinic acid itself if given in amounts exceeding the individual's tolerance (usually 50 to 100 milligrams by mouth) causes a pharmacological reaction – flushing, burning and tingling, and redness of the skin, due to peripheral vaso-dilatation; these reactions are, however, transient, and unpleasant rather than serious. Nicotinic acid amide (nicotinamide), on the other hand, does not cause these symptoms, and the mild toxic symptoms are considered to be due to the inability of the body to change the free acid into the amide with sufficient rapidity when large doses are given. In severe cases, especially when associated with vomiting, nicotinic acid amide should be given parenterally, either subcutaneously or intravenously.

In the vast majority of cases, treatment with nicotinic acid relieves the acute symptoms of pellagra promptly, and brings about a progressive return to normal even in severe cases. But treatment should not be confined to the administration of nicotinic acid. We have seen that pellagra is usually complicated by other deficiencies besides that of nicotinic acid, and, as in all deficiency diseases, specific therapy should be accompanied by the administration of a complete and adequate diet as soon as the patient is able to eat. Yeast and yeast extracts, being rich sources of all the vitamins of the B_2 complex, are valuable additions to the therapeutic diet. Milk, although a poor source of nicotinic acid itself, is a valuable adjunct in the treatment of pellagra, possibly on account of its rich riboflavin content or because it promotes the development of intestinal bacteria that synthesize nicotinic acid.

Nicotinic Acid in other Conditions. Nicotinic acid has been stated by Manson-Bahr (1941) to exert considerable beneficial effect in the treatment of sprue, but confirmation of this claim is needed as most of his patients were at the same time receiving parenteral injections of crude liver extract.

Natural Sources. Nicotinic acid is widely distributed in plant and animal foods, but only in relatively small amounts, except in meat (especially the organs), fish, wholemeal cereals and pulses In a normal Western European diet, about half the nicotini content is provided by meat and fish.

Nicotinic acid is readily soluble in water and is resistant to heat Cooking therefore causes little actual destruction of nicotinic acid but considerable amounts may be lost in the cooking water an 'drippings' from cooked meat if these are discarded. In an averag mixed diet, from 15 to 25 per cent of the nicotinic acid of th cooked foodstuffs may be lost in this way.

Commercial processing and storage of foodstuffs cause little los of nicotinic acid.

As a result of the wide distribution of nicotinic acid among foo stuffs and its stability to heat and storage, even a moderately goo diet should contain reasonable amounts of nicotinic acid, and tl occurrence of pellagra in endemic form is therefore an index of the inability of the population to secure a good mixed diet rather than a measure of the concentration of nicotinic acid in any particular foodstuffs. In this country, nicotinic acid deficiency, like aneurin and riboflavin deficiency, is only rarely seen, unless as a conditioned deficiency in diseases interfering with the absorption of or increasing the requirement of the vitamin.

Proprietary Preparations. Nicotinic acid tablets containing 50 milligrams of nicotinic acid are made by firms such as Allen & Hanbury, Bayer, Parke-Davis & Co., Burroughs Wellcome, Roche, and Crookes Laboratories. Ampoules containing 50 milligrams nicotinic acid for injection are produced by Burroughs Wellcome and Crookes Laboratories.

Nicotinamide – tablets or ampoules – containing 50 milligrams are produced by British Drug Houses and Roche.

Pyridoxin (*Vitamin B_6, Filtrate Factor I; rat anti-dermatitis vitamin*)

Chemical Nature. Pyridoxin was first identified about ten years ago by its biological activity in liver extract, from which aneurin and riboflavin had been removed. It was separated from such extracts by adsorption on to fuller's earth and was later identified chemically as a compound consisting of a pyridine nucleus to which hydroxyl, hydroxymethyl and methyl groups are attached.* Synthesis of the vitamin soon followed.

Pyridoxin is a white crystalline substance, soluble in water, and relatively resistant to heat.

Physiological Function. Pyridoxin has been shown to be an essential vitamin for the rat, the dog, the pig and the chick, but its function in the organism is unknown.

The structure of pyridoxin and the fact that it appears to be closely bound to protein (it is non-dialysable from yeast) suggest that it may, like riboflavin and nicotinic acid, exist in the cells in

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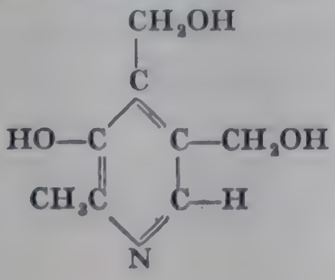
* Pyridoxin is 2-methyl-3-hydroxyl-4 : 5 hydroxymethyl pyridine, and has the following structural formula :



combination with protein as part of their oxidation-reduction system.

Pyridoxin has been shown to be a constituent of the co-enzyme of an enzyme concerned with the decarboxylation of certain amino-acids.

There appears to be some functional relationship between pyridoxin and the unsaturated fatty acids, since administration of the latter both protects against and cures the symptoms of pyridoxin deficiency in the rat. It has therefore been suggested that pyridoxin is in some way connected with the utilization of fatty acids, or the synthesis of fat from proteins.

Symptoms of Deficiency. As with other vitamins of the B complex, the symptoms of pyridoxin deficiency vary somewhat according to the species of the test animal. The most characteristic symptom is the dermatitis of the paws, ears and round the mouth of the rat, which is marked by swelling, œdema, redness and denudation, and is referred to as acrodynia. Other symptoms tha have been described in the rat are convulsions and a microcytic anæmia. In the pig and dog, the most prominent symptoms ar convulsions and a microcytic anæmia.

It is not yet known whether pyridoxin is essential in huma metabolism, and no clear-cut symptoms have been described in man Spies and his co-workers (1939) have, however, described a serie of residual symptoms in pellagrins treated by the addition aneurin, riboflavin, and nicotinic acid to their usual inadequat diets. These symptoms included great nervousness, irritability insomnia, weakness and difficulty in walking, and they responde dramatically to the administration of 50 milligrams of pure syn thetic pyridoxin. Some cases of cheilosis refractory to riboflav are stated to have responded to pyridoxin therapy.

Requirements and Dosage. The human requirement, if any, pyridoxin is unknown, but on the basis of animal experiments, has been deduced that it may be about 2 milligrams per day.

Natural Sources. Among the best sources are yeast, liver, ric bran, wholemeal cereals (about 0·5 mg. per 100 grams), me (0·4 to 0·7 mg. per 100 grams), and milk (0·2 mg. per 100 gram

Proprietary Preparations. Although there are as yet no defin indications for pyridoxin therapy in man, certain propriet preparations are available:

Pyridoxin (British Drug Houses) – 10 mg. per tablet, and mg. per ampoule.

Pyridoxin (Vitamins, Ltd.) – 10 mg. per tablet, and 50 mg. per ampoule.

Vitamin B_6 (Crookes Laboratories) – 10 mg. per tablet, and 50 mg. per ampoule.

Pantothenic Acid (*Chick Anti-dermatitis Factor; Filtrate Factor II*)

Chemical Nature. The vitamin activity of pantothenic acid, as of pyridoxin, was first demonstrated by fractionation of liver extract. The filtrate left after successive removal of thiamin, riboflavin and pyridoxin was found to be still effective in curing a dermatitis produced by feeding chicks on a basal ration deficient in the vitamin B_2 complex.

This 'filtrate factor' was later shown to be identical with a substance needed for the growth of yeast cells, and was given the name of pantothenic acid, on account of its presence, though in very minute amounts, in a wide variety of foodstuffs.*

Pantothenic acid has been synthesized and is usually prepared as the calcium salt, which forms fine white crystals. It is fairly stable to moist heat in neutral solutions, but is destroyed by prolonged dry heat or by hydrolysis in alkaline solution, which splits it into its two component acids.

Physiological Function. Although pantothenic acid appears to be an essential vitamin for certain animal species, such as the dog, the pig, the rat, the chick, its function in the body is unknown. There is evidence that it is present in animal tissues bound to protein, and it may therefore be a constituent of an enzyme system, like certain other members of the vitamin B_2 complex.

Symptoms of Deficiency. The vitamin activity of pantothenic acid is associated especially with the prevention of a peculiar dermatitis about the beak and eyes of the chick, but other species of animals develop lesions when deprived of pantothenic acid. In the rat, there is defective growth and necrosis of the adrenal cortex, accompanied by a dermatitis of the snout and a greying of the hair. Dogs deprived of pantothenic acid may show sudden collapse with

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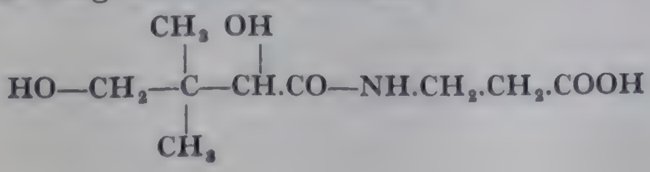
* Pantothenic acid is a compound formed of $\alpha:\gamma$-hydroxy-β-dimethyl butyric acid united by the peptide linkage to β-alanine. It has the following structural formula:



$$\mathrm{HO{-}CH_2{-}\overset{\displaystyle CH_3}{\underset{\displaystyle CH_3}{C}}{-}\overset{\displaystyle OH}{C}H.CO{-}NH.CH_2.CH_2.COOH}$$

biochemical changes in the blood similar to those of adrenal cortical deficiency, while in the pig there develops anorexia, diarrhœa and ulceration of the skin. Pantothenic acid is also a potent growth stimulator of yeasts and bacteria.

Its significance in human nutrition has not yet been established, but the fact that it is necessary for many species of animals suggests that it may be an essential part of every living cell and hence necessary for the nutrition of all the higher forms of life.

Requirements and Dosage. Since nothing is known as to the possible effects of pantothenic acid deficiency in man, no figures can be given for the daily requirement, although on the analogy of experiments with dogs, it has been claimed to be in the region of 5 milligrams per day.

Natural Sources. Liver is one of the richest natural sources, bu meat, wholemeal cereals and milk also contain good amounts. Th losses of pantothenic acid in cooking are possibly of the sam order as those of aneurin, which it resembles in its solubility an its destruction in alkaline solution.

Biotin (Vitamin H; 'Anti-Egg-White Injury Factor')

It has been known for many years that in experimental anima such as the rat, a diet containing a high proportion of raw eg white produces a characteristic dermatitis. These symptoms we prevented by boiling the egg-white before feeding, or by t addition of various foodstuffs to the raw egg-white diet, and it w at first considered that the skin lesions were due to a toxic fact in the raw egg-white. Investigation of the protective factor various foodstuffs led to its identification with a substance kno as biotin, which is one of the group of chemically-unrelated su stances that stimulate the growth of yeast. When the mic biological methods used to estimate biotin were applied to t constituents of the dermatitis-producing diet, it was found t this diet contained at least as much biotin as the control di It was therefore concluded that some substance in raw egg-w bound biotin and made it unavailable to the body. The compo in egg-white responsible for inactivating biotin has been identi as an albumin, to which the name avidin has been given.

Biotin has been isolated in crystalline form and has been sh to be a cyclic derivative of urea, with a ring nucleus contai nitrogen and sulphur, to which a fatty acid side-chain is attac It is a stable compound, being resistant to heat in acid solut but is inactivated by alkalis or by oxidizing agents.

Although biotin seems to be universally distributed among plant and animal tissues, its physiological functions, apart from its growth-stimulating properties for yeast, are quite unknown.

Deficiency of biotin can be produced in the chick, but not in other experimental animals, on a synthetic biotin-deficient diet, which suggests that, in most species, biotin can be synthesized by the intestinal bacteria in amounts sufficient to meet requirements, providing that it is not inactivated by adding egg-white to the diet.

One clinical case of possible biotin deficiency in man has so far been described (Williams, 1943), in a patient who had an abnormal liking for eggs, of which he consumed ten raw daily. He developed a scaly, exfoliative dermatitis confined almost entirely to the limbs. The skin lesion resembled histologically those of rats deprived of biotin, and improved steadily when the patient was given the normal hospital diet.

Choline

The nutritional importance of choline (trimethyl hydroxyethyl ammonium hydroxide), a component of the phospholipoid lecithin, is discussed in detail in Chapter XIII. The function of choline is in some way related to the mobilization of fatty acids in the body, and in its absence from the diet of experimental animals, accumulation of fat in the liver, hæmorrhagic necrosis of the kidneys and retardation of growth occur.

Because choline is necessary in the diet and because the lecithin-rich foodstuffs like meat, cereals and eggs are good sources of other members of the vitamin B_2 complex, choline is sometimes classified as a member of this complex.

Para-aminobenzoic Acid

Attention has been directed to the nutritional significance of this simple derivative of benzoic acid by studies on the mode of action of the sulphonamide drugs. Their bacteriostatic action has been shown to be due to interference with the metabolism of bacteria, but this property can be prevented by the addition of yeast or tissue extracts. The anti-sulphonamide factor of yeast when isolated was found to be para-aminobenzoic acid, a substance which had previously been identified as one of the factors necessary for the growth of bacteria. It has therefore been concluded that para-aminobenzoic acid is a component of an enzyme necessary for growth, and that the sulphonamides act by displacing para-aminobenzoic acid from this enzyme.

In the rat, deficiency of para-aminobenzoic acid has been claimed to induce greying of the fur, which is curable by administration of the acid in small amounts, but the recognition of para-aminobenzoic acid as a vitamin within the accepted meaning of this term and its significance in human and animal nutrition must await further investigation.

It is possible that it is not needed directly by mammals, but that its presence in ingested foodstuffs is of advantage in that it stimulates the growth of micro-organisms in the alimentary tract and so provides the host with other essential metabolites made by the micro-organisms.

Inositol

Inositol (hexahydroxy-hexahydrobenzene) is found in both plant and animal tissues. In plants, it occurs as the calcium magnesium salt of inositol phosphoric acid (known as phytin), and it is found in such animal tissues as brain, muscle, red blood cells, and the eye. In its pure state, inositol is a crystalline substance with a swee taste, and is an extremely stable compound, resisting treatmen with strong acids and alkalis.

Because of its vitamin-like action in certain species of animal and because it forms one of the group of substances which appear to stimulate the growth of certain bacteria, inositol has bee tentatively placed among the members of the vitamin B_2 comple In the rat, a deficiency of inositol appears to retard growth an cause alopecia, features that can be cured by its administratio it has also been found to prevent the development of fatty live in rats on a diet deficient in the vitamin B_2 complex.

The possible significance of inositol in human nutrition is as y unknown.

Folic Acid

Rats fed on a purified diet supplemented with adequate amou of all synthetic vitamins and given sulphaguanidine at the sa time develop leucopenia which can be prevented by the administ tion of liver extract. Monkeys, too, when fed on a purified di with added vitamins, develop a similar condition with loss weight, a moderate anæmia and a leucopenia, even when sulphaguanidine is given, and this syndrome also responds to li extract. Apparently the purified diet fortified with suppleme of the known vitamins lacks some factor, which can, howe be elaborated by the intestinal bacteria of the rat unless th organisms are destroyed by sulphaguanidine.

The factor which prevents the development of leucopenia on the synthetic diet is found in yeast, green vegetables and grass as well as in liver, and has been termed 'folic acid' from its presence in grass.

Folic acid concentrates have been found effective in the treatment of the leucopenia produced in rats and monkeys by the means described above, which suggests that folic acid may yet find a useful place in the treatment of the leucopenia that sometimes follows the administration of sulphonamides in man.

Until recently, folic acid preparations were confined to concentrates prepared from the foodstuffs rich in the vitamin. Within the last year, however, the synthesis of folic acid has been achieved by Angier and his co-workers (1945). The empirical formula $C_{15}H_{15}O_8N_5$ has been ascribed to it, and while no details regarding its structural formula have so far been published, folic acid appears to contain xanthopterin, a substance related to the purines, and itself of nutritional significance in that it prevents the development of anæmia in young rats fed on goat's milk.

Synthetic folic acid has been tested clinically by Spies and his collaborators (1946) in the treatment of macrocytic anæmias. They have found that the administration of synthetic folic acid, either by mouth or parenterally, is followed by prompt hæmopoietic response in cases of Addisonian pernicious anæmia, nutritional macrocytic anæmia, and the macrocytic anæmia of pregnancy, pellagra, and sprue. Associated with the hæmatological response is a striking clinical improvement, marked by increasing vigour and appetite.

The initial experiments of Spies have indicated that the minimal effective dose of synthetic folic acid by mouth is in the region of 10 mg. per day, but doses of as much as 400 mg. by mouth have been given without ill effect. Spies has suggested tentatively that the therapeutic dose should be 20 mg. daily by mouth or parenterally.

It is apparent therefore that folic acid is a potent anti-anæmic factor in certain types of macrocytic anæmia. The question whether it is identical with the anti-pernicious anæmia factor hitherto prepared by the chemical fractionation of liver must await further investigation.

Other Factors of the B_2 Complex

By the use of different species of test animals, evidence has been obtained of the existence of other factors of the vitamin B_2 complex besides those already described – for instance, the two other growth factors for the chick, termed the R and S factors, which have been obtained from yeast.

The Anti-anæmic Factor

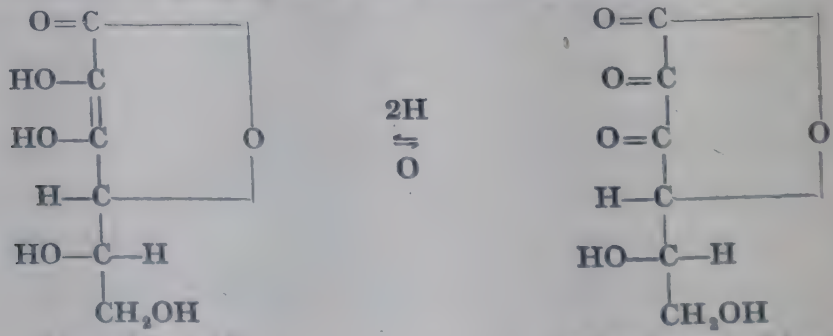
The greatest interest from the clinical point of view lies in the attempt to identify the anti-anæmic principle, and also the 'extrinsic factor' of Castle, which has been widely accepted for years as being associated with the vitamin B_2 complex, a conclusion that is supported by feeding experiments in animals. Thus, monkeys fed on a diet deficient in the vitamin B_2 complex and low in protein develop a macrocytic anæmia which is the analogue of tropical macrocytic anæmia and the macrocytic anæmia of sprue and is curable with yeast extracts, and pigs fed on a diet deficient in the vitamin B_2 complex develop a disease almost identical with pernicious anæmia in man, including the neurological disturbances, achlorhydria, and stomatitis. At one time, it was believed tha the extrinsic factor was identical with riboflavin, but this ha been disproved, and indeed tests of the anti-anæmic effect in pernicious anæmia of all the hitherto identified components of th vitamin B_2 complex either alone or incubated with gastric juic have given negative results. In view of the recent work on fol acid referred to above, it may yet be established that folic aci and the anti-pernicious anæmia factor are identical.

Vitamin C (Ascorbic Acid, Cevitamic Acid)

Chemical Nature

Ascorbic acid is one of the hexuronic acids, which are related the mono-saccharides.* It has been isolated and synthesized

* Ascorbic acid ($C_6H_8O_6$) has the following structural formula:



l-ascorbic acid. Dehydro-ascorbic acid.

Two stereo-isomeric forms exist, *l*-ascorbic acid and *d*-ascorbic a but only the *l*-form is physiologically active.

Ascorbic acid is a powerful reducing agent. The first stage o oxidation, to dehydro-ascorbic acid, is reversible: the relation between the two substances is shown above. Dehydro-ascorbic ac

ADDENDUM

Folic Acid

The chemical formula of synthetic folic acid and methods for its production have recently been published by Angier and his co-workers (1946). It is a derivative of pterin, a substance related to the purines, and is: N-[-{[(2-amino-4-hydroxy-6-pteridyl) methyl] amino} benzoyl] glutamic acid. Since folic acid can be replaced by much larger amounts of thymine (2,4-dioxy-5-methyl pyrimidine) as a growth factor for lactic acid bacilli, the former may possibly act as an enzyme or co-enzyme in the synthesis of thymine-like compounds which are utilised in the production of cell nucleic acid. Results similar to those reported by Spies in various types of megaloblastic anaemias (see p. 67) have been confirmed by other workers including the senior author (Davidson and Girdwood, 1946). It appears probable that further experience will show that it is in the treatment of the group of steatorrhoeas, which has hitherto been relatively unsatisfactory, that folic acid may find its principal place in therapeutics (see p. 383). Davis and Davidson (1944) and Davidson and Girdwood (1946) have shown that cases of megaloblastic anaemia refractory to the parenteral injection of potent chemically purified liver extracts will respond both to liver previously digested with papain (" proteolysed liver ") and to folic acid given orally. These workers produced evidence to show that the active principle in proteolysed liver was not folic acid, and suggested that liver contains other haematinic principles as yet unidentified in addition to folic acid and the specific anti-anaemic factor present in chemically purified liver extract. The hope that folic acid would be effective in the treatment of ideopathic agranulocytosis and of the leukopaenia that follows the administration of such drugs as sulphonamides and thiouracil in man has unfortunately not been fulfilled at the time of writing (see p. 66).

Pyridoxin

The successful treatment of agranulocytosis with pyridoxin (see p. 61) has been reported by Cantor and Scott (1945).

Casein Hydrolysates

In addition to the product mentioned on p. 389, the following improved casein hydrolysates of British manufacture, also largely free of undesirable by-effects, are now available: Casydrol (Genatosan), and Pronutrin (Herts. Pharmaceutical Co.).

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pure crystalline form as a white substance, readily soluble in water, of which the dry crystals are stable to air and daylight. In alkaline solution, ascorbic acid is very sensitive to oxidation, which is accelerated by heat and light, and by traces of metals, especially copper. In acid solution in the absence of metals, however, it is remarkably stable.

Physiological Functions

Although a great deal is known of the chemical and biochemical properties of ascorbic acid, little is known of its physiological functions and still less of the means by which these are fulfilled. In spite of its known activity as an oxidation-reduction agent *in vitro*, there is no evidence that ascorbic acid acts as a respiratory catalyst.

Animal experiments have shown that ascorbic acid is necessary for the proper formation and normal structure of the intercellular ground substance of mesodermal tissues such as connective tissue, bone, teeth and capillaries. Fibrils of material known as collagen form bundles in the intercellular ground substance under normal conditions. Collagen appears to be formed by 'jellation' of colloids in the intercellular ground substance, and ascorbic acid evidently controls this process, since in ascorbic acid deficiency collagen formation ceases, the intercellular ground substance loses its normal character, and the collagen bundles disappear.

Ascorbic acid is present in all body tissues, but is found in unusually high concentration in certain organs and tissues – the adrenal glands, the pituitary gland, and the intestinal wall.

It is rapidly absorbed from the intestine and is found in the blood and other body fluids. The white blood cells contain higher concentrations of ascorbic acid than the plasma. The concentration in the plasma reflects the recent level of ascorbic acid intake rather than the general level of ascorbic acid in the tissues, which is thought to be indicated more by the amount of ascorbic acid in the leucocytes. There does not seem to be any true storage of ascorbic acid in the tissues, since experiments have shown that when guinea-pigs are placed on a scorbutic diet the onset of scurvy is not delayed by previous saturation of the tissues and body fluids with ascorbic acid.

Excretion of ascorbic acid in the urine is variable, the amount depending on the ascorbic acid content of the blood and tissues

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readily reduced in the body to the original state, and is thus as potent an antiscorbutic as ascorbic acid itself. Oxidation beyond the dehydro form is irreversible, and destroys its antiscorbutic properties.

and hence on the recent dietary intake of ascorbic acid. Even when a subject has been on a vitamin C-free diet for some time, the urine contains material which reduces indophenol. However, this is probably not ascorbic acid.

Effects of Deficiency

The fundamental lesion of vitamin C deficiency is the defective formation of intercellular ground substance. This is a general process throughout the body, but the characteristic gross lesions occur in the bones, teeth, gums and capillaries.

(*a*) *Bones*. The skeletal lesions are most marked during the period of growth and are among the most constant features of scurvy in children, in whom bone growth becomes retarded and finally stops. The changes are general throughout the skeleton, but the bones most often and most severely affected are the lower end of the femur, the upper ends of the tibia and humerus, and th costochondral junctions, where widening of the epiphysis occurs similar clinically to that seen in rickets.

In the epiphyseal regions of the affected bones, the epiphysea cartilage loses its normal architecture and comes to consist o irregular masses of calcified cartilage embedded in fibrous tissue and a zone of fibrous tissue separates the calcifying epiphysea cartilage from the already formed bone of the shaft. The osteo blasts lose their shape and cease to form new bone, but bon resorption continues, so that there is a general rarefaction of th bones and an increased tendency to fractures.

(*b*) *Teeth*. In adults, the normal dentine is replaced by a inferior, less differentiated tissue, referred to as osteodentin Hyperæmia and œdema of the pulp develop, being followed b atrophy and degeneration of the odontoblast layer. The cementu shows changes similar to that occurring in the dentine, and th teeth become loose as a result of rarefaction of the alveol processes. These changes are reversible by treatment, but hist logical evidence of the defect always persists.

Although the pathological changes in the teeth of childr suffering from scurvy have not been extensively studied, it probable that similar lesions to those found in the teeth of adu occur and lead to defective formation of the teeth.

(*c*) *Gums*. The gums are affected only when there are tee The lesion begins with hyperæmia and redness of the interden papillæ, followed by swelling, hæmorrhage, retraction, ulcerati and secondary infection. Unlike the mouth lesions found deficiency of various factors of the vitamin B complex, scorbutic lesions of the mouth are strictly limited to the gums.

(*d*) *Capillaries.* It is possible that the defect occurring in the capillary walls in scurvy is due to loss of intercellular ground substance and collagen fibrils in the thin connective tissue around the capillaries, but there is no histological evidence for this view. As a result of the defect in the capillary walls, hæmorrhages occur in many parts of the body; those in the skin are characteristically petechial, perifollicular hæmorrhages, but large ecchymoses, intramuscular hæmorrhages, and hæmorrhages into serous cavities may also occur.

Vitamin C and Wound Healing

The pathological lesions described above are those of advanced vitamin C deficiency. But milder degrees of deficiency cause structural and functional changes, although these are not accompanied by gross lesions. Reparative processes, especially those involving connective tissue, are interfered with, and comparatively mild grades of vitamin C deficiency are said to interfere with the normal healing of wounds.

It can readily be demonstrated that there is considerable delay in the healing of wounds in scorbutic guinea-pigs and weakness of the scar tissue formed. This finding is true also of man, for in the case of experimental human scurvy, studied by Crandon and his co-workers (1940), there was no interference with wound healing after three months on a vitamin C-free diet, but after six months there was failure in wound healing, due to defective formation of intercellular substance and capillaries, which responded to several days' intensive vitamin therapy. Observations on wound healing in surgical patients have confirmed Crandon's findings, and suggest that there may be an increased need for vitamin C in the surgical patient, due to utilization of the vitamin in the process of tissue repair.

These results emphasize strongly the need for maintaining a normal state of vitamin C nutrition in surgical patients. But it must be pointed out that defective wound healing has only been demonstrated in persons known to have been depleted of vitamin C; and judging by animal experiments, and the observations of Fox and his co-workers (1940) in man, administration of vitamin C to subjects with normal vitamin C nutrition, or even with a low vitamin C intake where this has not led to actual signs of deficiency, will not influence the rate of wound healing.

Vitamin C and Infection

The patient with scurvy easily develops intercurrent infection. There is a good deal of evidence which suggests that vitamin C deficiency short of scurvy may predispose towards infective disease and prolong the period of illness. Studies both in experimental animals and in man have shown, too, that the vitamin C requirements are considerably increased in infections.

Vitamin C and Anæmia

It has long been held that vitamin C is one of the factors influencing the maturation of the red blood cells. This view has been based on animal experiments in which the anæmia and atrophy of the bone marrow that often develop in scorbutic guinea-pigs were found to respond to injections of vitamin C, and also on the frequent occurrence of anæmia in patients suffering from scurvy and the reports of several workers that this anæmia responded to vitamin C therapy alone.

The clinical evidence is, however, conflicting. Mild vitamin C deficiency is not associated with anæmia, and in the experimentally-induced case of scurvy reported by Crandon (1940), anæmia did not develop, although a leucopenia, cured by vitamin C administration, did appear. It is possible that when anæmia is associated with vitamin C deficiency it may be due to the consumption of a diet lacking in hæmopoietic factors as well as in vitamin C. Since, however, it has been shown that the addition of such factors – *e.g.* iron, liver-extract – may fail to cure the anæmia without the previous or simultaneous administration of vitamin C, it may be concluded that ascorbic acid is essential for normal hæmopoiesis.

Symptoms of Deficiency

(*a*) *In Adults.* Cases of frank scurvy in adults are rare in Britain, apart from occasional cases among indigent persons, usually men, living alone on a grossly unbalanced diet (the so-called 'bachelor's' scurvy), or in patients suffering from peptic ulcer or other gastro-intestinal disorder who have been kept for long periods on a too rigid milk diet.

Although the disease may have an apparently rapid onset, i requires a prolonged deficiency of vitamin C extending over severa months before frank scurvy develops. The appearance of the characteristic disease is preceded by a period of vague and in definite symptoms such as weakness, loss of weight, vague pain in the limbs, malaise, irritability or depression, and sometimes a mild anæmia. Where the deficiency in vitamin C is not pronounced, the disease may not progress beyond this stage, which is sometimes referred to as 'sub-clinical' scurvy.

The classical studies of Crandon, Lund and Dill (1940) have shown that in a previously well-nourished man consuming a diet devoid of vitamin C but otherwise adequate, the first symptoms suggestive of scurvy, a feeling of fatigue and weakness, take about three months to develop, while definite signs like petechial hæmorrhages and follicular hyperkeratosis do not appear until after four or five months.

If the deficiency is severe and prolonged, lassitude and weakness become more pronounced, muscle and joint pains occur, and signs appear relating to the hæmorrhagic tendency that is the essential manifestation of scurvy. This hæmorrhagic tendency is usually marked first by the appearance of petechial hæmorrhages in the skin round the hair follicles, but in severe cases more massive hæmorrhages occur in the muscles, or under the periosteum causing swelling and tenderness of the limbs, or into the serous cavities, or from the nose and the gastro-intestinal or urinary tracts.

Gingivitis of a characteristic type is a very common symptom of scurvy. The gums become bluish, swollen and spongy, this change occurring first in the interdental papillæ which become hyperplastic and tend to grow over the teeth, forming the so-called 'scurvy buds'; spontaneous hæmorrhages occur, and infection and ulceration of the gums develop. Trauma evidently plays a large part in localizing these gum lesions, since they only occur where teeth are present, and then largely at sites of irritation and stress such as are formed by tartar or badly spaced teeth. Due to the degenerative changes in the cementum and alveolar bone, the teeth become loosened and may fall out.

Much controversy has of late centred round the question whether the relatively common disease of the gums known as ulcerative gingivitis is in fact a manifestation of scurvy. Like the gingivitis found in scurvy, it occurs at sites of irritation and stress, and is associated with bleeding, ulceration and infection. The commonest organisms found together in the lesions are Vincent's fusiform bacillus and a spirochæte, but there is reason to believe that they are secondary invaders. Several workers have claimed that patients suffering from ulcerative gingivitis show evidence of vitamin C deficiency when subjected to the special chemical tests described below, and that the rate of healing of the gum lesions is accelerated by the administration of large doses of vitamin C in addition to the usual local treatment for the condition. On the other hand, many carefully controlled observations have failed to confirm such benefit from vitamin C administration. The weight of evidence in our opinion suggests that this disease is not related to vitamin C deficiency, but is of infective origin (possibly due to a primary virus infection), local trauma being a predisposing cause.

A skin lesion of great importance in the diagnosis of scurvy is the development of follicular hyperkeratotic papules, over the buttocks and lower limbs in particular. This has been ascribed by some observers to a complicating vitamin A deficiency, but it is of interest to note that in the case of experimental human scurvy described by Crandon and his co-workers follicular hyperkeratosis was the first physical abnormality to develop.

(*b*) *In Infants.* Infantile scurvy is the commonest form of this disease seen in Western Europe and America. There are no essential differences between scurvy in infancy and in adult life, the description of infantile scurvy as a separate disease (Barlow's disease or scurvy-rickets) being due to the confusion caused by the frequent combination of scurvy and rickets in the same infant at a time when the significance of deficiency diseases and the importance of their prophylaxis in infancy were not fully appreciated. The only differences are due to the effects of vitamin C deficiency on growing, as opposed to adult tissues, and relate chiefly to the bones. In infants suffering from scurvy, general symptoms such as pallor, weakness, irritability and often fever, are accompanied by signs referable chiefly to the long bones. There is pain and swelling of the lower limbs, usually at the lower ends of the thighs, due to intramuscular or subperiosteal hæmorrhages, and the infant lies in a characteristic attitude with the lower limbs everted and slightly flexed and shows disinclination to move the limbs. The swellings of the thighs are extremely tender on palpation and any attempt to handle the limbs causes crying.

The ends of the long bones often show enlargement like that seen in rickets, and epiphyseal separations or even fractures may occur. Radiologically, appearances due to the pathological changes described above may be detected – 'ground-glass' atrophy especially at the ends of the shafts, with indistinct trabecular markings, cortical atrophy, increased width and density of the zone of provisional calcification at the epiphyseal ends of the long bones and a zone of rarefaction in the shaft next to the zone of provisional calcification.

There are usually some petechial skin lesions in areas of pressure, although these are less common than in adults. Gingivitis only occurs if the teeth have erupted.

Special Tests for Vitamin C Deficiency

Although the symptoms and signs of scurvy described above may appear to develop quickly, they are in fact the effects of a severe and prolonged vitamin C deficiency. It is therefore very desirable that this deficiency should be recognized before the stage of clinical scurvy is reached. Unfortunately the early symptoms and signs of vitamin C deficiency are few and non-specific, and this, along with the comparative simplicity of the estimation of vitamin C in biological material, has led to the development of numerous special tests for vitamin C deficiency. But the very multiplicity of these tests and the conflicting views of the different workers as to their significance show that no fully satisfactory method for the diagnosis of vitamin C deficiency at present exists. These tests are, however, so commonly used, that a brief description of them, their scope, and limitations is necessary in any account of vitamin C deficiency and its diagnosis.

The tests fall into four main groups:

1. *Capillary Resistance Tests.* These were devised before the chemical procedures for the estimation of vitamin C were available. We have seen that a hæmorrhagic tendency, presumably due to increased capillary fragility, is an essential feature of scurvy. The rationale of the capillary resistance test is to make this hæmorrhagic tendency manifest before it appears spontaneously, by putting additional strain on the walls of the skin capillaries either by positive pressure induced with a tourniquet or by negative pressure applied with a suction apparatus. The capillary resistance test therefore can be classed as a functional test of vitamin C deficiency in that it purports to be a measure of an increased capillary fragility caused by a *pathological* degree of vitamin C depletion.

Unfortunately, neither the degree of pressure used nor the time of its application is uniform in the methods used by different workers, and there are other drawbacks to the test, of which the most serious is that it is by no means always positive, even in the presence of clinical scurvy. For instance, Macmillan and Inglis (1944), in a recent survey of fifty-three cases of clinical scurvy, found that in forty patients tested by a positive pressure method before and after saturation with vitamin C, only twenty-one gave a positive result before and a negative result after saturation, six gave a positive result before and after saturation, while no less than thirteen gave a negative result throughout.

Therefore, while a positive response in the absence of other causes of increased capillary fragility may be suggestive of scurvy, a negative response by no means excludes that diagnosis.

2. *Urinary Excretion Tests.* Vitamin C can be estimated in urine and other biological fluids by titrating against a solution of the blue dye dichlorphenolindophenol, which is reduced and decolorized by ascorbic acid.

Testing of single specimens of urine for their vitamin C content is of little value, but it has been claimed that a fair indication of the state of vitamin C nutrition can be obtained by measuring the amount of vitamin C excreted in a twenty-four-hour specimen of urine. There is a wide variation in the amount of vitamin C excreted in the urine, both between different individuals, and at different times in the same subject. It is claimed, however, by the advocates of this test that there is a lower level below which the daily excretion of ascorbic acid in a normal person should never fall. This level is about 15 milligrams of ascorbic acid per day in an adult and perhaps 10 milligrams per day in a child. If the daily urinary excretion of vitamin C is being used as a test of deficiency, repeated estimations are necessary.

3. *Saturation Tests.* These tests depend on the observation that in subjects who have lived on a diet low in vitamin C for some time, administration of a large dose of ascorbic acid causes a smaller and slower rise in the level of ascorbic acid in the plasma than in a normal individual. The administered vitamin is quickly removed from the blood stream into the other body fluids and tissues, and as a result little or none of the administered vitamin is excreted in the urine. If the same large dose of ascorbic acid is administered on successive days, there is a steady rise in the fasting plasma level towards the threshold value for urinary excretion (about 1·4 milligrams per 100 c.c.), but the vitamin continues to be removed quickly from the blood and hence does not appear in the urine until the body fluids and tissues are saturated with vitamin C. When this point is reached, the administered ascorbi acid now ceases to be removed rapidly from the blood stream int the tissues, but instead is excreted, so that the amount of ascorb acid in the urine shows a sudden marked increase to a high lev

which is maintained if dosage with ascorbic acid is continued. The attainment of this high level is interpreted as indicating saturation of the body with vitamin C.

If the plasma vitamin C level or the amount of vitamin C in the urine is measured, either in the twenty-four-hour specimens or in the urine collected during the period of maximum excretion following administration of the test doses (about 4 to 6 hours afterwards if the dose is taken by mouth), the day on which saturation is reached can be gauged, and the degree of 'unsaturation' of the subject expressed either as the number of days or the amount of ascorbic acid administered before saturation is reached. Unfortunately, variations in the technique employed by different workers and the interpretation of their results make it difficult or impossible to compare the various procedures that have been advocated for the carrying out of the saturation test.

The saturation test as applied to the urinary excretion of vitamin C was originally devised by Harris and Ray (1935) as a measure of the state of vitamin C nutrition, and has been extensively employed in the United Kingdom. It has also been the subject of much criticism, chiefly on the grounds that a high degree of 'unsaturation' may be found in the absence of any symptoms of scurvy. Judging by the few studies of experimental human scurvy that have been carried out, it is reasonable to assume that, on a diet devoid of vitamin C, a high degree of 'unsaturation' would be encountered weeks and even months before definite clinical signs of scurvy appeared. The saturation test cannot therefore be regarded as an aid to the diagnosis of scurvy, although an extreme degree of 'unsaturation' will of course be found in this disease. It is doubtful, too, if 'unsaturation' as measured by the saturation test can be used even as an indication of a degree of vitamin C deficiency that is near the scurvy level. In our opinion, the saturation test should be looked upon as a guide to the recent dietary intake of vitamin C, and as such it has a sphere of usefulness in assessing the vitamin C intakes of subjects at different times, or of different groups of people, when more direct methods of measurement are not available.

4. *Concentration of Vitamin C in the Blood.* The concentration of vitamin C in the plasma depends on the amount of vitamin C in the diet, the vitamin C content of the tissues, and the excretion of the vitamin by the kidneys. Consequently, it varies greatly from individual to individual, and in the same individual at different times, chiefly as a result of differences in the vitamin C intake.

The concentration of vitamin C in the plasma in well-nourished persons whose tissues are saturated with the vitamin ranges from about 0·8 milligram per 100 c.c. to over 1·0 milligram per 100 c.c. Various workers have suggested different levels of plasma ascorbic acid, usually in the neighbourhood of 0·3 milligram per 100 c.c. and lower, as indicating a state of serious vitamin C deficiency. But it is extremely doubtful if it is justifiable to place this interpretation on low plasma ascorbic acid levels. Thus in the recent studies of human experimental scurvy already referred to, the plasma ascorbic acid level fell to 0·1 milligram per 100 c.c. only nine days after the subject began to eat a vitamin C-free diet and reached zero level in six weeks, although clinical signs of scurvy did not develop for another thirteen weeks. The plasma ascorbic acid level seems therefore to be an index of the recent dietary intake of vitamin C rather than of the state of vitamin C nutrition of the tissues, and this is borne out by the results of numerous surveys of groups of individuals in normal health in whom a not-inconsiderable proportion can usually be found with plasma ascorbic acid levels below 0·3 milligram per 100 c.c.

The ascorbic acid content of the leucocytes is several times greater than that of the plasma – the average normal concentration is about 35 milligrams per 100 grams, with a range of 29 to 43 milligrams. Unlike the plasma vitamin C level, the ascorbic acid content of the leucocytes falls only slowly when the diet is devoid of vitamin C and reaches zero level only shortly before the appearance of clinical scurvy. It reflects the state of vitamin C nutrition of the tissues more closely than any of the other tests described, and while it cannot be regarded as an index of the recent vitamin C intake or early depletion, estimation of the vitamin C content of the leucocytes furnishes a guide as to how protracted the period of depletion has been and to the imminence of clinica scurvy.

Requirements and Dosage

It must be admitted that in the present state of knowledge, n definite figure or range of figures can be quoted as the daily vitami C requirements of man, and the subject has been the centre of muc controversy. Arguments about man's optimum vitamin C requir ments tend to crystallize around two opposing views, one claimi that full saturation of the body tissues and fluids represents th optimal state of vitamin C nutrition and the other that not on freedom from all symptoms and signs of scurvy but also a co pletely healthy condition can be maintained without saturation of the tissues and fluids with vitamin C.

The argument that full saturation of tissues and body fluids is necessary for optimal vitamin C nutrition is based on the following:

(*a*) the observation that in animals such as the rat, which can synthesize their own vitamin C, saturation of tissues and body fluids is the rule, the same being true of the guinea-pig (one of the few species other than man that cannot synthesize vitamin C), if given a free choice of diet.

(*b*) the claim made by several workers that wound healing is accelerated if ascorbic acid is taken in amounts sufficient to raise the vitamin C content of the plasma to a 'saturation' level of 1 milligram per 100 c.c. or over.

A daily intake of 50 to 100 milligrams of ascorbic acid is required to maintain not only tissue saturation but also a plasma vitamin C concentration of about 1 milligram per 100 c.c.; and it is on this basis that the figure of 75 milligrams has been accepted by many workers, especially in America, as the optimum daily vitamin C requirement of an adult male.

These opinions have been challenged, chiefly in Great Britain, on both experimental and clinical grounds. Thus Zilva (1936) found that the minimum daily dose of vitamin C required to prevent macroscopic and microscopic lesions of scurvy in the guinea-pig was 2 milligrams, whereas 5 to 8 milligrams daily were required before detectable amounts of ascorbic acid were found in the tissues and 20 milligrams daily before full saturation of the tissues was attained. In a series of experiments in man, Kellie and Zilva (1939) found that 30 to 40 milligrams of ascorbic acid daily were required to maintain saturation of the tissues although not of the body fluids. On the assumption that the dose of ascorbic acid per unit of body weight is the same in man as in the guinea-pig, Kellie and Zilva (1939) argued that 3 to 4 milligrams daily (*i.e.* one-tenth of the saturation dose) would be the minimum dose to prevent even microscopic lesions of scurvy, and that 10 to 15 milligrams should be sufficient to produce a slight but significant deposition of ascorbic acid in the tissues and maintain an individual in good health, and they concluded that this figure cannot be far removed from the actual requirement of man.

On the other hand, however, in a large-scale study carried out by Fox and his co-workers (1940) on South African miners, twelve cases of scurvy occurred among the control group taking a diet computed to contain 12 to 25 milligrams of ascorbic acid per day, whereas only one case, and that a mild one, occurred in a group receiving a supplement of 40 milligrams daily in the form of an orange-juice concentrate. Apart from these cases of scurvy, however, no difference was found in physical endurance, healing of wounds, or the condition of the gums. It is perhaps unjustifiable to place too much stress on this variation in the incidence of scurvy in the two groups, as personal idiosyncrasies in the diet of the men developing scurvy may have been a factor. But it suggests that an intake of 10 to 15 milligrams of ascorbic acid daily for an adult is more nearly a minimum than an adequate one. At the same time, it is known that many persons probably receive only about these amounts of vitamin C in their diet and apparently remain in good health, and also that, as mentioned already, a large proportion of apparently healthy individuals have plasma ascorbic acid levels that supporters of the 'saturation' hypothesis would consider as evidence of vitamin C deficiency.

While these findings make it difficult to believe that unsaturation of the tissues with vitamin C necessarily denotes a state o deficiency, we have come to the conclusion, after weighing up th evidence, that the desirable daily intake in an adult and in childre other than infants is in the region of 30 milligrams daily, th amount required to maintain the tissues but not necessarily the bod fluids in a state of saturation. This intake can readily be achieved without recourse to specially rich sources of vitamin C such a citrus fruits, by taking one big or two smaller servings of potatoe and vegetables daily, provided that these have been cooked i such a way as to avoid undue destruction of ascorbic acid. O the other hand, it requires more careful selection of the diet t attain intakes of vitamin C as high as 75 milligrams daily an this we consider unnecessary, except perhaps in conditions wher the needs for vitamin C are enhanced, such as pregnanc and lactation and illnesses associated with fever or heightene metabolism.

Treatment of established scurvy demands a larger intake vitamin C of course. In mild cases, a diet specially chosen for i high vitamin C content and containing anything up to 200 mill grams of ascorbic acid daily, secured by the inclusion of rich sourc of vitamin C such as citrus fruits and tomato, is all that is require In severe cases, the high vitamin C diet of the type mention above should be supplemented by oral administration of t vitamin in amounts up to 500 milligrams or even more dai

In infants suffering from scurvy, about 100 milligrams daily is probably sufficient.

Natural Sources

Vitamin C has a more limited distribution than the other important water-soluble vitamins. Citrus fruits, currants and berries, and green vegetables are the richest sources among the most commonly consumed plant foods. Root vegetables and potatoes contain smaller amounts, but the latter, although containing a relatively low concentration, are certainly not a negligible source. On the contrary, potatoes, on account of the amount normally eaten every day, account for a considerable proportion of the total vitamin C intake of the population of Great Britain and other European countries, especially in the lower economic groups who cannot afford to buy much fruit. They were particularly important during the War, when the import of citrus fruits to all intents and purposes ceased. Except for liver and other glandular tissues, and the roe of certain fishes, foodstuffs of animal origin contain at best only traces of ascorbic acid. These animal sources cannot, however, be considered as negligible, since they suffice to protect races like the Eskimos, whose diet consists almost solely of meat, fish and fat.

Stability and Effects of Cooking

Vitamin C is the least stable of the vitamins, being very readily oxidized to biologically inactive substances through the activity of an enzyme, ascorbic acid oxidase, present in plant tissue, or by the direct action of heat. Both these factors operate to reduce significantly the amount actually consumed in vegetables as compared with the amount present in the growing plant. In the case of green vegetables, the vitamin C content begins to fall as soon as they are garnered; and after storage for a few days at ordinary temperature, it may have fallen to about half what it was in the growing plant. Bruising and bad storage methods, such as keeping vegetables tightly packed in sacks, aggravate this loss. Loss of vitamin C also begins as soon as potatoes are lifted, and about three-quarters disappears during storage in the clamps during the winter.

In cooking vegetables, the vitamin C content is reduced both by actual destruction through enzyme action and heat and by extraction of the vitamin into the cooking water. The instability of ascorbic acid in the cooking of vegetables has perhaps been somewhat exaggerated in the past, but it is nevertheless true that adequate retention of the vitamin can only be obtained by the employment of reasonable care in cooking.

The greater part of the loss from green vegetables is due to solution of the vitamin in the cooking water. There is not a great deal of actual destruction of the vitamin, for heat rapidly penetrates the loose-textured vegetable tissue, destroying the ascorbic acid oxidase. Prolonged cooking, however, leads to considerable loss from direct heat oxidation. The extraction of vitamin C into the cooking water occurs in the first few minutes of boiling, so that this source of loss can only be minimized by keeping the volume of cooking water small (but not so small that it goes of the boil when the vegetables are added) and by ensuring that i is not thrown away but is used for stock.

Steaming green vegetables, if carried out very carefully, doe lead to greater retention of vitamin C than boiling, but the los of vitamin is due to actual destruction rather than extractio and increases progressively and rapidly with the time of steaming In general, it may be said that boiling vegetables is mor satisfactory from the point of view of vitamin retention tha steaming. Keeping vegetables hot after they are cooked is i essence steaming, and is just as destructive of ascorbic acid. It i safe to say that this bad catering practice is responsible for mor avoidable loss of vitamin C than any other single cooking factor

The points that must be observed to secure maximum retentio of vitamin C in green vegetables have been summarized as follow by the Accessory Food Factors Committee of the Medical Researc Council (1941):

(1) Use when fresh.
(2) Cool, damp storage.
(3) Avoidance of crushing or bruising.
(4) Use of the smallest possible quantity of boiling water t which salt (2 teaspoonfuls to the pint) has been added.
(5) The reduction of the cooking time to the minimum necessar to make the foodstuff tender.
(6) Dishing up and serving as soon as possible after cooking.
(7) Cooking water should be drained off and used for soups an gravies, or for cooking of successive batches on the san day.

Owing to the more solid texture of root vegetables and potato much less ascorbic acid is dissolved out into the cooking wat than from green vegetables. But the more solid texture preven such rapid heat penetration, and so allows more time for the ascorbic acid oxidase to continue its activity before being destroyed by heat. As a result, more of the vitamin C lost in boiling root vegetables is actually destroyed than is dissolved out into the cooking water. Boiling potatoes in their skins reduces the already small loss through extraction of vitamin C into the cooking water, but does not affect the greater loss through actual oxidation of the vitamin. The practice does have one definite advantage, however, when the saving of food is an urgent necessity – for after boiling, the skin can be peeled off cleanly, thus saving the edible tissue lost in the peeling of raw potatoes.

As in the case of green vegetables, keeping root vegetables and potatoes hot after cooking causes a progressive destruction of the vitamin C left after boiling. Mashing the vegetables exposes a greater surface to the air and so aggravates the destruction of vitamin C.

Canned vegetables and fruits are popularly considered as of little value as sources of vitamin C, but if modern canning methods are used, in which care is taken to eliminate factors tending to destroy vitamin C, canned products may have an even higher vitamin C content than the corresponding domestically-cooked fresh foodstuffs.

Proprietary Preparations

Synthetic ascorbic acid preparations in tablet form or in solution for injection are produced under a variety of trade names.

(*a*) Tablets of 5, 25 or 50 milligrams ascorbic acid (Burroughs Wellcome, Allen and Hanbury, B.D.H.); ‘Cantan’ (Bayer); ‘Planavit C’ (May & Baker); ‘Redoxon’ (Roche); Vitamin C (Crookes Labs., Parke Davis & Co.); ‘Davitamon C’ (Organon); ‘Celin’ (Glaxo); ‘Vitamin C Pabyrn’ (Paines and Byrne).

(*b*) Ampoules containing 100 milligrams or 500 milligrams in solution for injection: Ascorbic Acid (Allen & Hanbury, B.D.H.); ‘Cantan’ (Bayer); ‘Celin’ (Glaxo); ‘Davitamon C’ (Organon); ‘Redoxon’ (Roche); ‘Vitamin C Pabyrn’ (Paines & Byrne).

Vitamin P.

Chemical Nature

The existence of this vitamin was first reported in 1936 by the Hungarian scientist Szent-Györgi, who claimed that extracts of paprika and citrus fruit juices were more effective than pure vitamin C in the treatment of patients suffering from increased vascular permeability, as measured by capillary fragility tests. The same worker later reported the isolation from these extracts of an active preparation consisting of a mixture of two flavonone glycosides, hesperidin and a glycoside of eriodictyol. The flavonones are water-soluble yellow plant pigments, which occur naturally as glycosides combined with glucose or rhamnose as the carbohydrate part of the molecule, and are found in relatively high concentration in citrus fruits, rose hips and black currants. The exact chemica identity of vitamin P is still a matter of some doubt, however

Physiological Function

Practically nothing is known of the effect of vitamin P in th normal subject, as all the evidence so far obtained for its activit relates to its effect in experimental animals and subjects considered to be deficient in the vitamin.

Effects of Deficiency

Szent-Györgi claimed that vitamin P decreased the number hæmorrhages in scorbutic guinea-pigs and prolonged the surviv period. The latter finding has not been confirmed, but oth workers have brought forward evidence in support of the vie that vitamin P does decrease the capillary fragility of scorbut guinea-pigs. Bacharach and his co-workers (1942) have used th effect as a test method for assessing the amount of vitamin P foodstuffs. They found that animals receiving a scurvy-producin diet show a steady decrease in capillary resistance even when give adequate amounts of ascorbic acid, and that this fall is abolish by hesperidin and many fruits and vegetables and extracts pr pared from them.

Clinical studies suggest that this vitamin is also essential human nutrition. Increased capillary fragility has been fou frequently in patients suffering from multiple vitamin deficienci and citrus juices have been shown to be effective in increasi capillary resistance, even when pure vitamin C had failed produce a response.

Scarborough (1940) whose studies have contributed much of t evidence relating to the human need for vitamin P, has suggest that at least two forms of subcutaneous bleeding may occur the result of nutritional deficiency in man, one due to vitamin deficiency and the other to vitamin P deficiency. The former characterized by the large subcutaneous and intramuscular hæmorrhages seen in scurvy, in the treatment of which vitamin P is ineffective, and the latter occurs in the form of petechial hæmorrhages into the skin, often around the hair follicles. He states that vitamin P can increase the capillary resistance of scorbutic subjects, and that vitamin P deficiency may exist even after treatment with ascorbic acid. The characteristic manifestations of vitamin P deficiency, according to Scarborough, are weakness, lassitude, pains in the shoulders and in the legs after exertion, and a marked decrease in capillary resistance, which is followed by the development of the petechial hæmorrhages as described above, especially in areas exposed to pressure. These manifestations respond to treatment with vitamin P. Thus, while the symptoms and signs mentioned above are held by most authorities to be characteristic of scurvy, Scarborough's work suggests that the clinical syndrome of scurvy is due to a double deficiency, namely of vitamins C and P, and that the administration of rich sources of both vitamins such as citrus juices or rose hip syrup offers a more rational therapeutic approach than the prescription of synthetic preparations of the individual vitamins.

When considering the treatment of purpura it must be remembered that conditions other than vitamin P deficiency may lead to a lowering of capillary resistance. For instance, in idiopathic thrombocytopænic purpura or in purpura associated with blood dyscrasias such as leukæmia and aplastic anæmia, no benefit could be expected to result from the administration of vitamin P. On the other hand, reports have appeared of the successful treatment of vascular purpuras (of the non-thrombocytopænic type) with preparations of vitamin P, and vitamin P has been claimed to be effective in counteracting the increased capillary fragility occurring as a toxic manifestation of antisyphilitic arsenic therapy. Some writers have reported successful results in the treatment of hæmorrhagic (focal) nephritis and other forms of hæmaturia with vitamin P, although others have been unable to reproduce these results.

In view of these equivocal findings, it is probably wise at the present time for the practitioner to prescribe vitamin P only for cases of purpura believed to be due to a dietary deficiency, the vitamin being administered either in the form of citrus juices or rose hip syrup, or as the proprietary preparation 'Permidin' (Glaxo), available in tablets containing 0·15 gram of vitamin P. A total dose of 1 gram should be given daily.

THE FAT-SOLUBLE VITAMINS

Vitamin A

Chemical Nature

Vitamin A is a complex monohydric alcohol,* consisting of a ring nucleus (the beta-ionone nucleus) to which an aliphatic side chair is attached. The vitamin is formed in the animal body from certai

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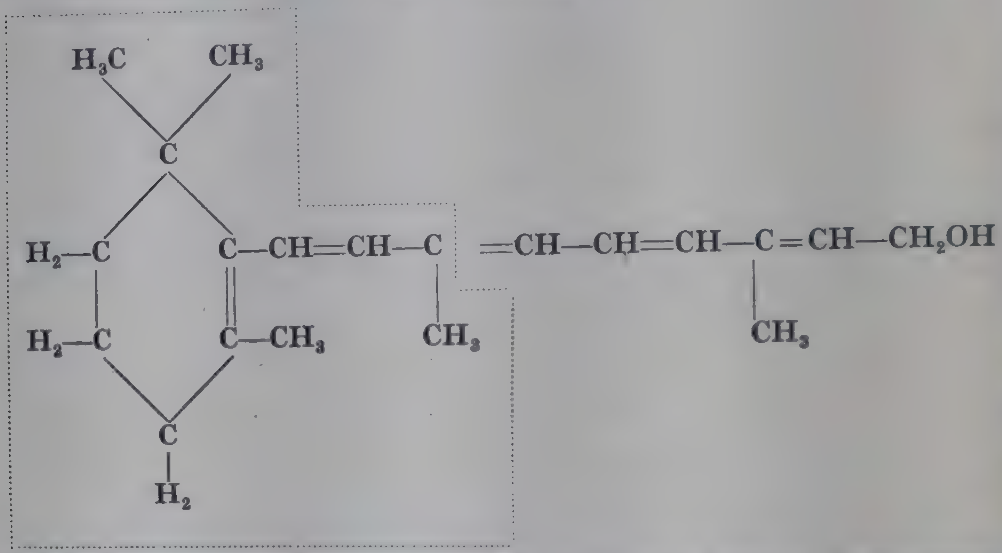
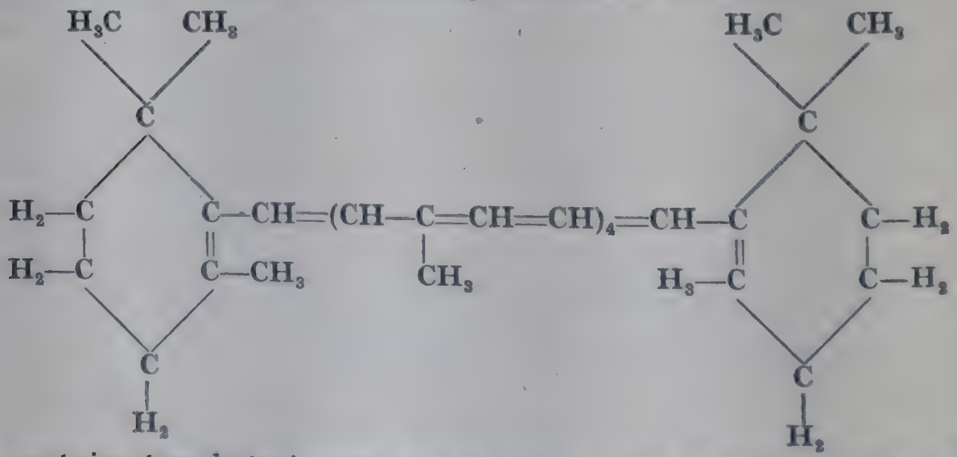
* The structural formula of vitamin A is –



The beta-ionone nucleus (enclosed in the dotted lines) is an essenti component of the vitamin A molecule, and only carotenes containin this nucleus can be converted into vitamin A in the body.

Synthesis of pure vitamin A has been achieved.

Beta-carotene has the following structural formula –



It contains two beta-ionone nuclei and if the beta-carotene molecu is split in two by hydrolysis at the middle of the aliphatic cha linking these two nuclei, two molecules of vitamin A would be forme In the body, however, the beta-carotene molecule evidently does n undergo this symmetrical splitting, and only one of the beta-iono nuclei is utilized in the formation of one molecule of vitamin A, sin weight for weight beta-carotene is only half as active biologically vitamin A.

yellow or red plant pigments known as carotenes, four of which are important in animal nutrition: alpha-, beta-, and gamma-carotene, and cryptoxanthine. Beta-carotene is the most abundant of the carotenoid pigments in the common plant foods and therefore the most important of the precursors of vitamin A.

Animals cannot synthesize vitamin A and therefore obtain it either by the conversion of carotene obtained from plant foods or by eating animal tissues in which vitamin A has been stored.

Physiological Function

Carotene is converted to vitamin A in the liver by the action of an enzyme known as carotenase, but the two substances are by no means identical from the point of view of biological activity, carotene being neither absorbed nor utilized so readily in the body as preformed vitamin A contained in animal foods.

The presence of bile in the intestine is essential for the absorption of carotene, possibly because carotene is absorbed in the form of a water-diffusible compound with bile acids. Fat in the diet is also important for the good absorption of carotene, probably because it dissolves carotene and so facilitates absorption. Bile, however, is apparently unnecessary for the absorption of vitamin A, although fat in the diet evidently aids absorption. Vitamin A occurs naturally in combination with fatty acids as an ester, which is hydrolized in the intestine like the other fatty-acid esters of the food. Vitamin A is absorbed in the form of the alcohol, which is recombined with fatty acid to the ester form before passing into the lacteals; this absorption is rapid and complete under normal conditions.

On the other hand, the absorption of carotene from the intestine is far from complete, and unless the diet is very deficient in vitamin A, even the amount absorbed is not completely utilized. There is considerable variation in the ability of different animal species to absorb and utilize carotene. Therefore, although the International Unit of vitamin A is defined as the amount which has the same biological activity as 0·6 microgram of pure beta-carotene, the vitamin A potency of a plant food is not necessarily indicated by its carotene content. In man it would appear that the ratio of absorption of carotene and its conversion into vitamin A varies according to the type of plant food – thus about half the carotene of green leafy vegetables appears to be utilized, whereas the proportion is much smaller in the case of the carotene contained in carrots. In Great Britain it has been accepted that about one-third of the carotene contained in the plant foods of an ordinary mixed diet is utilized as vitamin A. On this basis, therefore, the amount of carotene in plant food equivalent in biological activity, as far as man is concerned, to one International Unit of preformed vitamin A would be 1·8 micrograms not the 0·6 microgram of the International Standard.

After absorption, vitamin A and carotene are carried to the liver, where the carotene is converted into vitamin A, and the vitamin A is stored. This store is lowest at birth and increases with age, th amount stored depending on the dietary intake of vitamin A an carotene. Where the intake has been adequate, this reserve o vitamin A is sufficient to tide the subject over a considerable perio of lowered dietary intake, but where a low intake has been customary, the reserve is correspondingly low, and a sudden drop in intak may precipitate the onset of deficiency symptoms. Factors othe than age affect the storage of vitamin A, for low liver reserves hav been found in many diseases which either decrease its absorptio (*e.g.* entero-colitis and cœliac disease), hinder the conversion o carotene into vitamin A, and impair the storage capacity of th liver (*e.g.* hepatic disease, and chronic nephritis, where the toxæmi appears to interfere with the conversion of carotene), or increas excretion by the kidney. Vitamin A is never excreted by th kidneys in health, but it may appear in the urine in illness especially in pneumonia and to a lesser extent in chronic nephritis

The fundamental part played by vitamin A in the metaboli processes of the body is unknown. The relationship of the requir ments of vitamin A to body weight rather than to metaboli activity suggests, however, that the vitamin is essential for cellula structure rather than function.

Effects of Deficiency

Studies of the effects of deficiency of vitamin A show that it ha the following important functions:

(*a*) Vitamin A is essential for normal growth. This is of cours a non-specific function since deprivation of any vitamin other factor essential for normal nutrition will arrest growt The growth effect of vitamin A is probably related to i influence on the growth of bone; thus, in young rats on vitamin A-deficient diet, the skeleton is the first body tiss to show impairment of growth.

(*b*) The most specific tissue change associated with vitamin deficiency is a metaplasia of various epithelia, in which t specialized epithelium is replaced by stratified squamous keratinizing epithelium. This suggests that vitamin A is necessary for the full evolution of certain highly specialized epithelial structures. The changes produced in different epithelial tissues are characterized by atrophic changes in the specialized epithelial cells, which is followed by replacement through proliferation of the basal cells to form a stratified squamous epithelium, over which the specialized epithelium sloughs off. Glandular structures whose epithelium is affected diminish their secretions, and the ducts are plugged with masses of cornified cells, the latter manifestation usually preceding the involvement of the acinar cells so that retention cysts occur in the glands. These changes occur in most of the epithelial tissues, such as the respiratory and genito-urinary tracts, eyes, skin, and the enamel of the teeth.

The developing teeth both in young animals and in infants are prone to severe damage in vitamin A deficiency, since the enamel organ, which is of epithelial origin, undergoes the characteristic atrophic changes and keratinizing metaplasia. The odontoblasts, although of mesodermal origin, are secondarily affected by the degeneration of the enamel organ and form defective dentine. Vitamin A appears to be the most important vitamin for the normal development of both enamel and dentine, and the tooth lesions due to vitamin A deficiency are hence the most important dental effect of any of the vitamin deficiencies. Probably the commonest sign of vitamin deficiency in man is the defective dentition arising as a result of an insufficient intake of vitamin A. The most important result of the metaplasia of epithelial surfaces described above is a lowering of the local resistance to infection of the epithelial tissues affected, so that in experimental animals, and probably also in man, vitamin A deficiency is associated with an increased incidence of infections involving epithelial surfaces. But the general defence mechanisms of the body are not impaired in vitamin A deficiency, and the vitamin is therefore ‘ anti-infective ’ only in the sense that, in subjects suffering from vitamin A deficiency, administration of the vitamin will increase the power of epithelial surfaces to resist local infection by restoring them to their correct and normal structure. The popular tendency to refer to vitamin A as the ‘anti-infective vitamin’ is thus correct only to a limited extent, the vitamin having no effect on the resistance to infection in subjects who have not been on a vitamin A deficient diet long enough to develop epithelial metaplasia.

(*c*) Vitamin A is an essential constituent of the retinal pigment found in the rods of the retina and known as visual purple.

The rods and cones of the retina are not directly stimulated by light, and the process of vision is much like that in the production of a photograph in a camera. The lens of the eye focuses the image on the retina, and photochemical processes occurring in the retinal pigments under the influence of light produce the stimulus to the optic nerve that is translated into vision by the brain. When light rays impinge on visual purple, it is bleached to a yellow pigment called retinene, which is related to carotene. Retinene cannot respond to light until it is changed back again to visual purple, and this regeneration is accomplished by the addition of vitamin A and protein. Part of the vitamin A for this reaction is supplied by retinene itself, but the reversible reaction between visual purple and retinene is not completely efficient, and the eye must receive from the blood a constant supply of vitamin A to supplement that derived from retinene. If this supply is not maintained to the optimum extent, the rate o regeneration of visual purple is delayed, and hence, afte exposure the eye takes longer to adapt itself to the receptio of new images. Vision in a bright light is carried out by th cones, which are probably not affected by lack of vitamin A whereas vision in a dim light is the function of the rods. I a bright light, bleaching of visual purple occurs, so that th retina contains little. When a person moves from a brigh light to a dim light, the rods cannot function properly unti the bleached visual purple is regenerated, and hence vision i momentarily impossible until regeneration of visual purpl begins, after which the eye becomes progressively adapted t vision in a dim light. If vitamin A deficiency is present, thi process of dark adaptation will be delayed, and since, eve in a dim light, some destruction of visual purple occur severe degrees of vitamin A deficiency may prevent sufficien visual purple ever being formed to provide normal nigh vision, so that complete night blindness results.

(*d*) Several workers have found degenerative lesions of the per pheral nerves, the optic and auditory nerves, and the sensor tracts of the medulla and spinal cord in animals suffering from vitamin A deficiency. According to Irving and Richards (1940), degeneration in the medulla of young rats can be produced with such certainty by deprivation of vitamin A that this procedure may be used as a method of assaying vitamin A.

Other workers, however, have failed to produce degeneration of the nervous system even in the presence of severe signs of vitamin A deficiency. The view is widely held that the degenerative lesions of the nervous system which have been described are due to compression of nervous tissue, occurring as a secondary effect of other lesions produced in young animals by vitamin A deficiency. Such compression may be due to an actual overgrowth of the bones of the skull, as demonstrated by Mellanby (1941) in vitamin A deficient puppies, or to a more pronounced inhibition of skeletal than of neural growth in vitamin A deficiency, with the result that the central nervous system outgrows its bony case and nerve roots herniate into the cranial and spinal foramina. The failure of some workers to find signs of nerve degeneration in vitamin A deficiency may thus be explained by the use of adult animals in their experiments.

In man, neurological symptoms are not found in association with signs of vitamin A deficiency, except in the disease known as *lathyrism* – an upper motor neurone paralysis affecting chiefly the lower limbs. Lathyrism occurs only under famine conditions, and is thought to be due to the action of toxins present in vetches on a nervous system whose resistance to these toxins has been weakened by a poor diet. Vitamin A deficiency has been singled out among the effects of a poor diet as being responsible for the debility of the nervous system, owing to the frequency with which night blindness is found among populations afflicted with lathyrism and the statements of some workers that vitamin A exerts a protective effect.

(*e*) Vitamin A deficiency has been suggested as an ætiological factor in the formation of renal calculi in man, following the observation that in experimental animals deprived of vitamin A the shedding of cells into the urinary tract from the keratinizing metaplastic epithelium of the renal pelves, ureters, and bladder gives rise to foci round which salts are deposited to form calculi. It has been claimed, too, that cases of renal calculi in man are frequently, though not invariably, associated with defective dark adaptation.

In spite of this evidence, however, the consensus of opinion is that vitamin A deficiency is not a factor of importance in the formation of urinary calculi in man.

Recent work has suggested another relationship between vitamin A deficiency and the urinary system. Animals suffering from this deficiency have been found to have an abnormally low urea clearance, not associated with any structural changes in the kidneys. Conversely, administration of excessive amounts of vitamin A temporarily raises the urea clearance above normal, this latter observation having been repeated in man. Although it is too early to assess the significance of these results, they are at least suggestive of a purely functional effect of vitamin A on the renal epithelium.

Symptoms and signs of Vitamin A Deficiency in Man

These originate as a result of impairment of two of the most important functions of vitamin A – the preservation of normal night vision and the maintenance of the specialized structure of epithelial surfaces.

(*a*) *Night Blindness*

A defect in the dark adaptation of the eyes is generally the first symptom to develop, the severity varying with the degree of the deficiency. In the mildest degrees of deficiency, the subject may be unaware of the defect, which can only be detected by special diagnostic methods; but when the deficiency is more severe, the patient may complain voluntarily of difficulty in vision at night, *e.g.* in finding a seat in a darkened hall, stumbling in the dark, or trouble in driving a car at night.

Detection of night blindness by measuring the rapidity of dark adaptation with a special photometer has therefore been widely used as a method of diagnosis of the earlier stages of vitamin A deficiency. The principle of the method is to bleach the visual purple of the retina by exposing the eyes to a bright light for a given time. The subject is then placed in the dark, and is shown small test flashes of light, whose brightness is increased until they are visible to him. The minimum amount of illumination required to make the light flashes visible is taken as a measure of the regeneration of the subject's visual purple. A single reading may be taken at the end of a standard period of time in the dark (usually ten minutes, as dark adaptation improves so rapidly up to this time that it is difficult to get readings), or the rate of regeneration of visual purple may be estimated by taking successive readings at intervals of a few minutes.

Defective dark adaptation may be due to causes other than vitamin A deficiency such as congenital night blindness, retinitis pigmentosa, and neurosis. But there is fairly general agreement that if due regard is paid to these other possible causes, impairment of dark adaptation can be accepted as a sign of early vitamin A deficiency. This view is based on the observations that defective dark adaptation is commoner in persons consuming a diet low in vitamin A than in those on an adequate diet; that it can be produced experimentally in man by feeding a diet deficient in vitamin A; that it can be cured by giving vitamin A; and that nutritional night blindness is usually associated with a low level of vitamin A in the plasma.

Certain workers have, however, dissented from this view, their objections being based chiefly on the lack of definite correlation between the state of dark adaptation and the level of vitamin A in the plasma, and on the failure of some investigators to produce a deterioration in dark adaptation in persons fed on a diet deficient in vitamin A. Variations in the amount of vitamin A stored in the liver of the experimental subjects may perhaps have been a factor in producing these apparently discordant results.

The observation that administration of orange juice along with vitamin A enhanced its effect in improving defective dark adaptation led to the suggestion that vitamin C is important in dark adaptation as well as vitamin A. But further investigation has shown that this effect of orange juice is not due to ascorbic acid, and it has been suggested that, in addition to an inadequate vitamin A intake, a simultaneous deficiency of some hitherto unidentified factor present in fresh fruits and vegetables plays a part in the production of defective dark adaptation.

(*b*) *Xerosis Conjunctivæ and Xerophthalmia*

Metaplasia of the conjunctival epithelium, which stops the secretion of its mucous cells, together with a reduction in the flow of tears due to obstruction of the ducts of the lacrimal glands by metaplastic changes in their lining epithelium, cause drying of the conjunctiva: if the eyelids are held open for a few moments, lack of lustre of the cornea and conjunctiva due to these causes can readily be detected. Later, when definite xerosis conjunctivæ has developed, the conjunctiva becomes greasy in appearance and is thrown into vertical wrinkles, most conspicuous over the temporal half. An increase in melanin pigment, giving rise to an irregular brown pigmentation of the conjunctiva, has been described frequently as occurring in vitamin A-deficient subjects belonging to dark-skinned races, to which this sign is possibly confined.

Heaping up of the keratinized epithelium in patches leads to the formation of what are known as *Bitot's spots* – small but conspicuous white plaques on the surface of the sclera which look like flakes of whitewash, almost always found on the temporal half of the conjunctiva. When xerosis conjunctivæ has reached this stage, secondary infection is liable to occur, leading to photophobia and inflammation, with a sticky conjunctival discharge. It is at this stage that the most serious effect of vitamin A deficiency may supervene, for the cornea may become œdematous, infiltrated with leucocytes and necrotic. These changes produce whiteness and opacity of the cornea, the condition being known as *keratomalacia.* Infection, ulceration, and finally perforation of the weakened cornea and destruction of the eye are the end results of this condition, which in its severest form is most common in infants and young children and is remarkable for its comparative painlessness and lack of photophobia, in contrast to corneal ulceration from other causes.

(*c*) *Phrynoderma*

The term 'phrynoderma' or 'toad skin' has been given to a skin eruption that is one of the characteristic signs associated with the keratinizing metaplasia of epithelial surfaces caused by vitamin A deficiency. There is a generalized hyperkeratosis of the epidermis, and the pilosebaceous follicles become plugged with masses of she cornified cells, which form horny plugs blocking the lumen an mouth of the follicles, whose sebaceous glands tend to atrophy The sweat glands do not undergo much change, but their ducts ar often blocked with keratinized material.

These structural changes in the epidermis and its appendage cause a dryness and roughness of the skin, which is followed by papular eruption about the hair follicles, most marked on th extensor surfaces of the arms and thighs, but extending in som cases to the back and abdomen. The papules are dry an contain a horny plug which either projects from the surface or covered by a loosely adherent scale. Unlike the eruption of acn the papules in phrynoderma practically never undergo pustulatio

There is still some doubt as to whether phrynoderma is the result of a vitamin A deficiency alone. Contradictory reports have been published as to the closeness of correlation noted between xerophthalmia and phrynoderma among populations where both conditions are relatively common, and more than one worker has pointed out that children appear to suffer from the eye lesion but not the skin eruption, whereas in adolescence and adult life the skin lesion is the more common. The increased incidence of phrynoderma after puberty may be related to the changes in the sebaceous glands and hair which occur at that time. Indeed it has been suggested that the mild follicular keratosis that is not uncommonly found in older children and adolescents in this country may be due to endocrine influences rather than to a nutritional deficiency. But the fact that some workers have cured cases of phrynoderma by the administration of vitamin A concentrates is in favour of the view that the underlying cause of this lesion is a vitamin A deficiency, although there may be additional factors, of nutritional or other origin, that render the skin more prone to the development of the lesion in the presence of a deficiency of vitamin A.

(d) *The Level of Vitamin A in the Blood*

Estimation of vitamin A in the blood is based on the blue colour that develops when vitamin A reacts with antimony trichloride. It is unfortunately too complicated a procedure to be used as a routine diagnostic method, but there is accumulating evidence that a low level of vitamin A in the blood is present along with defective dark adaptation as an early sign of vitamin A deficiency. The normal range of vitamin A in the blood serum is from 100 to 300 International Units per 100 c.c., and most workers agree that defective dark adaptation is always present when the blood vitamin A falls below 70 International Units per 100 c.c. Much lower values are found when signs of advanced vitamin A deficiency like xerophthalmia are present.

e) *Other Effects of Vitamin A Deficiency*

Although post-mortem evidence indicates that other epithelial surfaces than those of the skin and the eyes are affected in vitamin A deficiency in man, these lesions produce no characteristic clinical manifestations, except perhaps respiratory infections which appear to be frequent in the subjects of this deficiency. Metaplastic changes in the epithelium of the bronchioles lead to plugging of the lumen with masses of desquamated, cornified cells and the production of areas of atelectasis and bronchiectasis. Peribronchial pneumonia is thus a common complication of vitamin A deficiency in children.

Requirements and Dosage

In discussing the physiology of vitamin A (see pp. 87 to 88), it was pointed out that although the International Unit is defined as the amount which has the same biological activity as 0·6 microgram of pure beta-carotene, factors affecting the absorption and utilization of carotene in man make its biological activity much less than that of an equal amount of preformed vitamin A, and that on an average, the biological activity of plant foods can be represented in terms of preformed vitamin A if 1·8 micrograms of carotene, not the 0·6 microgram of the International Standard, are taken as equivalent in potency to one International Unit of preformed vitamin A. If this arbitrary assumption is made, the vitamin A potency of the carotene-containing foods of the diet can be added directly to that of foods containing preformed vitamin A and the total vitamin A potency of the diet thus expressed in terms of preformed vitamin A. In the following discussion the requirements and dosage of vitamin A will be stated only in terms of the preformed vitamin.

The human requirements have been assessed almost entirely by the estimation of the dose of vitamin that will just prevent the development of defective dark adaptation, or restore this to norma in subjects in whom defective dark adaptation has been induced by a diet deficient in vitamin A. The minimum amount of vitamin A per day that will prevent impairment of dark adaptation but provide little or no surplus for storage (*i.e.* the minimum for the maintenance of freedom from symptoms of deficiency under norma conditions) appears to be about 1,400 International Units for th 'average' man of 70 kilograms (11 stones). Since the requirement of vitamin A are dependent only on body weight and not on age an physical activity, the minimum requirement is perhaps bette stated as 20 International Units per kilogram of body weigh Other estimates have placed the minimum requirement as high a 2,000 International Units for an adult.

The ideal intake to be aimed at is of course not the minimu required to prevent symptoms of deficiency, but the amount th will provide enough of a surplus to permit the building up reserves as a safeguard against possible periods of decreased co sumption. The optimum is usually considered to be 3,000 Inte national Units for a normal adult, but some workers have set it at the higher level of 60 International Units per kilogram of body weight, or 4,200 International Units for an average man.

The vitamin A requirements are increased in pregnancy and lactation, since the body weight of the developing fœtus and infant must be added to that of the mother when assessing the requirement. The matter is dealt with in detail in Chapter IX, and all that need be said here is that estimates of the requirement vary from 4,000 to 6,000 International Units per day.

Any disease interfering with the absorption of vitamin A and carotene, the conversion of carotene into vitamin A, or the storage of vitamin A in the liver, will enhance the requirements of vitamin A. Among the more important diseases interfering with absorption of vitamin A and carotene are cœliac disease and conditions interfering with the entrance of bile into the intestine. Diabetes is the outstanding example of conditions that interfere with the conversion of carotene into vitamin A, while diseases of the liver interfere both with this function and with storage of vitamin A.

In established vitamin A deficiency, large doses of the vitamin are required to bring about a cure, and the administration of one or other of the concentrates described below is necessary along with the provision of a diet rich in vitamin A. Daily amounts of 20,000 to 40,000 International Units should be sufficient unless the deficiency is complicated by any condition interfering with absorption or storage. Night blindness responds rapidly to such doses, and recovery is a matter of days or at most a few weeks. Xerosis conjunctivæ also responds quickly, but xerophthalmia and phrynoderma require a longer period of therapy for their relief. The reason for this variation in response is doubtless because defective dark adaptation, at least in its earlier stages, is a functional disorder, whereas the severe eye and skin lesions are manifestations of structural damage to epithelial tissues and time is required for their anatomical repair.

Natural Sources

Preformed vitamin A is found in only a few foods, of which the most important are milk, butter, cheese, egg-yolk, liver, and some of the fatty fish. The liver oils of fish are the richest natural sources of vitamin A, but these are of course important as dietary supplements rather than as normal articles of diet.

Carotene is widely distributed among plant foods. It is found most abundantly in green vegetables (in which it is always associated with chlorophyll, so that while the green outer leaves of vegetables like cabbage and lettuce are rich in carotene, the white inner leaves contain little or none), and in the yellow vegetables and fruits.

All margarine manufactured for table use in Great Britain is now compulsorily fortified with vitamins A and D (see page 173).

Vitamin A and carotene are insoluble in water, and are stable at high temperatures in the absence of oxygen, although both are readily destroyed at all temperatures when air or oxygen is bubbled through solutions containing them. Ordinary cooking processes are therefore unlikely to cause much loss of either carotene or vitamin A, although the slow cooking of vegetables over long periods, as in stews, is said to cause some loss of carotene. It is stated that the vitamin A in butter is unaffected when the butter is used for frying, roasting or pastry-making. Recent work has shown that there is rapid and complete destruction of the carotene of red palm oil used to fortify cooking oil when the latter is raised to the temperatures used in frying, although this observation is of course of immediate importance only in tropical dietetics.

Preservation of vegetables and fruits by methods other than slow sun-drying does not affect their carotene content.

Official and Proprietary Preparations

For convenience these preparations are classified into two groups

(i) Preparations containing little or no vitamin D:

	International Units
Liquor Vitamini A Concentratus (B.P. Addendum 1940)	50,000 per c.c.
Avoleum (B.D.H.)	30,000 per gram 4,500 per capsule
Davitamon A (Organon)	6,000 and 60,000 per c.c 60,000 in 1 c.c. oil solution per ampoule
Essogen (Lever)	2,000 per capsule
Prepalin (Glaxo)	72,000 per c.c. 24,000 per capsule 100,000 in 1 c.c. per ampoule
Vitamin A (Crookes)	33,000 per capsule 100,000 in 1 c.c. per ampoule

(ii) Preparations containing vitamins A and D:

	International Units	
	A	D
Liquor Vitaminorum A et D Concentratus (B.P. Addendum 1940)	50,000 per c.c.	5,000 per c.c.
Halibut Liver Oil (B.P. Addendum 1940)	30,000 (min.) per c.c.	No standard
Cod-Liver Oil (B.P. Addendum 1936)	600 (min.) per c.c.	85 (min.) per c.c.
Oleum Vitaminatum (B.P. Addendum 1940)	1,000 per c.c.	100 per c.c.
Adexolin (Glaxo)	12,000 per c.c.	2,000 per c.c.
	4,500 per capsule	900 per capsule
Advita (Lever)	2,000 per capsule	200 per capsule
Davitamon A—D (Organon)	6,000 per c.c.	5,000 per c.c.
	3,000 per capsule	2,000 per capsule
Halibol (Allen & Hanbury)	40,000 per gram	6,500 per gram
	4,500 per capsule	900 per capsule
Haliverol (Parke, Davis & Co.)	27,000 per gram	5,400 per gram
	4,500 per capsule	900 per capsule
Radiostoleum (British Drug Houses)	15,000 per gram	3,000 per gram
	4,500 per capsule	900 per capsule
	75,000 per ampoule	15,000 per ampoule
Super D Oil (Crookes Labs.)	65,700 per gram	52,000 per gram
	11,000 per capsule	9,000 per capsule

Vitamin D

Chemical Nature

Some ten or eleven distinct but closely related compounds possessing antirachitic properties are known. All these are derived from sterols, complex substances of high molecular weight often closely associated with, but not related to, fats in plant and animal tissues. Certain of these sterols when exposed to ultra-violet irradiation undergo a change in their structure with the formation of new substances having antirachitic properties, this process being known as activation of the sterol. The activated sterols, though separate substances, are so closely related that many show no ordinary chemical differences, even in the usual structural formula. Only two of the activated sterols are of importance in nutrition and therapeutics: * (See note on following page.)

Vitamin D_2 (Calciferol)

Calciferol is made by exposing ergosterol (a sterol found in plants, especially in the lower orders like fungi and yeasts) to the action of ultra-violet light. Irradiation of ergosterol produces a series of reactions which give rise to several related substances, some toxic, of which only calciferol has a marked antirachitic action. In the manufacture of calciferol, irradiation has therefore to be controlled carefully, so that toxic products are not present, except in traces, in the irradiated ergosterol solution.

Although calciferol is widely used in therapeutics, it must be

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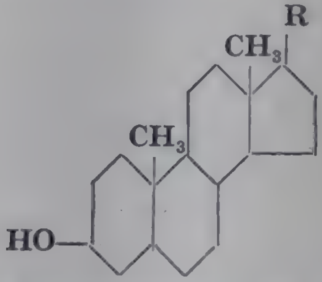
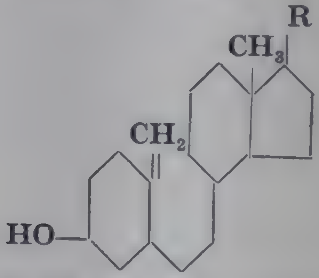
* All the compounds possessing antirachitic properties contain the sterol nucleus:



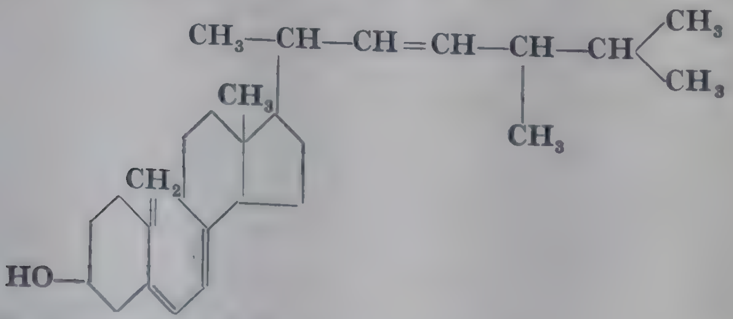
They differ, however, in the structure of the side-chain, representec by 'R' in the above formula.

Activation with ultra-violet light involves an opening of the ring between carbon atoms 9 and 10, viz.:

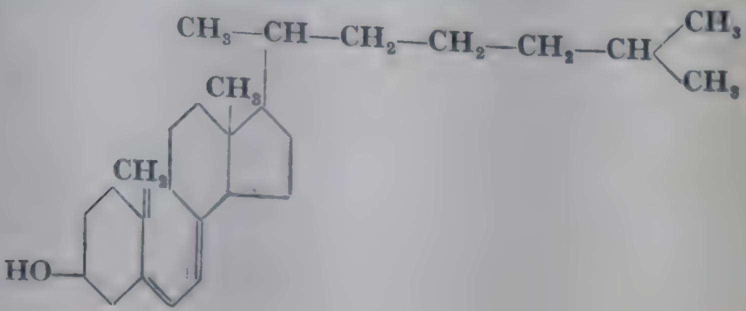


The structural formulæ of the two most important antirachiti substances are:

Vitamin D_2 (Calciferol, activated ergosterol):


CH3—CH—CH=CH—CH—CH
CH3
CH3
CH3
CH3
CH2
HO—

Vitamin D_3 (Activated 7-dehydrocholesterol)


CH3—CH—CH2—CH2—CH2—CH
CH3
CH3
CH3
CH2
HO—

emphasized that it is not a naturally-occurring vitamin, except for the relatively small amounts present in certain fish-liver oils. Ergosterol only occurs in plants, and animals can neither make it nor absorb it from their food. Irradiated ergosterol (calciferol) can be absorbed and utilized, but this is not significant in relation to natural diets, since living plants do not contain ergosterol in activated form.

Vitamin D_3

This substance is the natural vitamin D. It is formed by the irradiation of 7-dehydrocholesterol, a sterol widely distributed in animal fats, such as the oil of the preen glands in birds, and the oily secretions of animal and human skin. Activation of 7-dehydrocholesterol appears to take place on the surface of rather than in the skin, this being followed by absorption of the vitamin from the skin, or in animals by ingestion as a result of licking the fur.

The liver oils of fish are very rich in vitamin D_3, but it is not known how fish acquire this vitamin, unless by the ingestion of minute plants and animals (plankton) that live near the surface of the sea, and are hence exposed to solar irradiation.

Calciferol and vitamin D_3 have the same antirachitic potency for rats, but vitamin D_3 is considerably more potent for chickens. Evidence of a similar difference in potency in man has been adduced, but this is disputed by many workers.

Physiological Function

As mentioned above, the vitamin D formed by activation of 7-dehydrocholesterol in or on the skin is absorbed from the skin. Absorption of ingested vitamin D requires the presence of bile in the intestine.

Storage of vitamin D absorbed either from the skin or the intestine occurs in various tissues of the body for periods that can be measured in weeks. Depletion of these stores occurs partly by excretion of the vitamin into the small intestine and partly by its destruction in the body.

Although the effect of vitamin D in promoting normal calcification of bone is universally known, the exact site and fundamental nature of its activity are as yet uncertain.

Growth in length of the long bones of the body occurs at the band of epiphyseal cartilage lying between the shaft or diaphysis and the epiphysis. New cartilage is continuously formed on the epiphyseal aspect of the epiphyseal cartilage; while on the diaphyseal aspect, the cartilage degenerates and is invaded by capillaries and osteoblasts which form osteoid tissue in which bone salts are deposited. The formation of new cartilage keeps pace with its degeneration and the formation of new bone at the end of the diaphysis, so that the bone grows in length, growth ceasing when the epiphyseal cartilage ceases to form new cartilage and the diaphyseal bone meets and fuses with that of the epiphysis.

In vitamin D deficiency, the epiphyseal cartilage grows, but its degeneration becomes defective, so that there is widening o the zone of cartilage between diaphysis and epiphysis. On th diaphyseal aspect, the cartilage is irregularly invaded by capillaries and osteoblasts and osteoid tissue is formed, but n deposition of bone salts takes place. Calcification also ceases i the subperiosteal bone formation that produces thickening of th shaft, although increased amounts of osteoid tissue are forme there too.

Accompanying these changes in vitamin D deficiency are others which are probably more nearly associated with the fundamenta nature of vitamin D activity:

(*a*) Alteration in the levels of calcium and inorganic phosphoru in the serum. In animals the alteration varies with th amount of the two substances in the diet, but in man, th phosphorus is usually low, and the calcium normal, althoug the latter may be greatly reduced, when the phosphorus often normal.

(*b*) Increased phosphatase activity of the blood.

(*c*) Increased fæcal and a decreased urinary excretion calcium and phosphorus.

The site of action of vitamin D is evidently not the developin bones, for bones of animals suffering from vitamin D deficienc will calcify normally if placed in normal serum, and similar the injection of inorganic calcium and phosphorus salts in animals suffering from vitamin D deficiency will induce norm calcification.

The part played by vitamin D in calcification therefore appea to be to ensure that there is a sufficient supply of inorganic c cium and phosphorus in the fluids bathing the developing bo from which bone salts can be laid down. How vitamin D d this is as yet unknown, but it may be through the mobilizati of organic phosphorus compounds from the tissues, these bei converted into inorganic phosphorus by the bone phosphata The local concentration of inorganic phosphorus would thus raised, and by the law of mass action, this would lead to the precipitation of insoluble calcium phosphate from the serum. Vitamin D also increases the absorption of calcium from the gut, but whether this is a direct effect on calcium metabolism or is secondary to the action of vitamin D on phosphorus is not known.

Since vitamin D and the hormone of the parathyroid gland both have the effect of raising the level of the serum calcium, it was at one time thought that vitamin D produced its effects by stimulation of the parathyroid glands. It is now known, however, that vitamin D can raise the level of the serum calcium in animals and human beings suffering from parathyroid deficiency, although admittedly much larger doses are required than those that will correct vitamin D deficiency in normal subjects. Another argument against this mode of action of vitamin D is the difference in the action of the vitamin and the hormone. The hormone increases the urinary excretion of phosphorus, causing a lowering in the level of inorganic phosphorus in the serum followed by a rise in the serum calcium and an increased excretion of calcium in the urine, so that a negative balance of both calcium and phosphorus results; whereas vitamin D in anything but excessive doses keeps the serum calcium and phosphorus normal and promotes a positive balance of both elements. Indeed, Albright and Sulkowitch (1938) have brought forward evidence that part of the metabolic abnormality in rickets is actually an overactivity of the parathyroid glands, occurring as a compensation for the low blood calcium that results from defective absorption of calcium from the intestine. They suggest that it is this parathyroid overactivity which causes the low level of phosphorus in the blood which is one of the most characteristic findings in rickets, and that vitamin D, by promoting the absorption of calcium from the intestine, leads to a reduction in parathyroid activity and hence to a rise in the level of the blood phosphorus, so that normal deposition of bone salts can take place.

Excessive doses of vitamin D many times larger than those required for the treatment of deficiency of the vitamin, cause premature calcification of the cartilage and overactive bone formation with the result that the spongiosa becomes dense and compact. Metastatic calcification occurs in the soft tissues, especially the renal tubules and the tunica media of the large arteries, and the levels of calcium and phosphorus in the serum rise to abnormal heights. In experimental animals, loss of weight and death occur, and occasional fatal cases in children have been reported.

There is some doubt as to whether the symptoms of hypervitaminosis D are due to the vitamin itself or to toxic products formed along with calciferol during the irradiation of ergosterol – the former view is upheld by the fact that injection of excessive amounts of very pure calciferol preparations are toxic.

Symptoms of Deficiency

Lack of vitamin D, whether due to defective dietary intake or to inadequate exposure to sunlight, leads to a failure of calcification of developing bone, or where bone growth has ceased, to a decalcification of the bones and the formation of osteoid tissue under the periosteum. While the fundamental lesion is the same, the clinical manifestations vary according to the age of the subject. In infants and young children, vitamin D deficiency causes rickets, in adolescents, the condition is described as 'late rickets' or 'juvenile osteomalacia', while in adult life, the result of vitamin D deficiency is osteoporosis, or osteomalacia if the lesion is severe.

Rickets

In temperate climates, the lack of sun and the expense of foods rich in vitamin D, together with the failure to secure the consumption of vitamin D concentrates by nursing mothers and children in compensation, combine to make rickets a relatively common disease. It is certainly the commonest of the deficiency diseases, and estimates of its frequency in both the active and healed stages among children of the lower economic groups in England and America range from 25 to over 75 per cent.

While rickets can occur at any age, it is most frequently seen in infants between the ages of six months and two years.

The pathological changes occurring in growing bones as a result of vitamin D deficiency have already been described in the section dealing with 'Physiological Function'. But rickets is more than a disease of bone; it affects the whole body and produces symptoms other than those related to the skeleton. The infant with rickets is usually fat and to the superficial glance appears well nourished. But it is restless and fretful, pale, and with flabby muscles and relaxed ligaments, which allow the limbs to assume unnatural postures. Head sweating and head rolling are common, and the abdomen is distended, due to the flabby abdominal muscles, the atony of the intestinal musculature, and the intestinal fermentation arising from the excessive carbohydrate of the diet. Gastro-intestinal upsets with diarrhœa are common, and the infant is prone to respiratory infections, although this is probably due to the poor diet as a whole rather than to a deficiency of vitamin D alone. Development is delayed in that the teeth often erupt late, and there is failure to sit up, crawl, and walk at the normal ages.

The bony changes are of course the most characteristic and easily identifiable signs of rickets. The pathological changes in the bones produce extension and widening of the epiphyseal cartilages and a generalized defect in calcification. The earliest bony lesions to appear are craniotabes (small, round, unossified areas in the flat bones of the cranium, yielding like parchment, with a crackling feeling, to the pressure of the finger) and enlargement of the epiphyses especially at the lower ends of the radius and ulna, and of the costochondral junctions, the latter leading to the clinical sign known as beading of the ribs or the 'rickety rosary'. Craniotabes usually disappears before the end of the first year, but later evidence of rickets in the cranial bones is bossing of the frontal and parietal eminences due to overgrowth of osteoid tissue, and delayed closure of the anterior fontanelle. Deformities of the chest such as undue prominence of the sternum caused by the sucking in of the softened ribs on inspiration and Harrison's sulcus make their appearance.

If rickets persists into the second year of life, the bony signs described above persist or increase, and in addition new deformities develop, due to the added gravitational and muscular strains caused by the infant sitting up and crawling – kyphosis of the spine, and enlargement of the lower ends of the femur, tibia, and fibula. When the rachitic child begins to walk, deformities of the shafts of the leg bones become added to the list of lesions, and the spinal kyphosis is replaced by lordosis. Pelvic deformities are prone to develop at this stage, and in females later lead to serious difficulties at parturition. The radiological appearances of the long bones in established rickets are chiefly as follows:

(*a*) Rarefaction of the shaft with coarsening of the trabecular structure and blurring of the outlines due to the formation of osteoid tissue under the periosteum.

(*b*) Unevenness and raggedness of the end of the diaphysis with cupping.

An alarming complication is spasmophilia – the hyperexcitability of the nervous system in cases where a deficient calcium intake has led to a fall in the blood calcium. The symptoms may be carpo-pedal spasms, laryngismus stridulus, or generalized convulsions, the last being dangerous to life. Between attacks of any of these major manifestations, evidence of latent tetany – Chvostek's or Trousseau's signs – may be disclosed on examination.

Late Rickets or Juvenile Osteomalacia

While rickets is predominantly a disease of infancy and early childhood, it can occur at any age, especially during periods of rapid growth when the requirements for calcium, phosphorus and vitamin D are enhanced. Late rickets thus develops most frequently at the time of the second dentition and at puberty. The clinical manifestations are modified from those described above by the more advanced stage of development of the skeletal system, and consist chiefly of osteoporosis and deformities, especially genu valgum and spinal curvature. A deficiency of calcium in the diet is very commonly present as well and contributes to the pathological picture.

Osteomalacia

This disease is now recognized as the adult counterpart of rickets, but an inadequate calcium intake is probably always associated with a deficiency of vitamin D in its production. An inadequate supply of vitamin D lessens the proportion of available calcium that is absorbed from the food, but this can be compensated for, to a certain extent at least, by a liberal calcium intake. Hence, a deficiency of calcium in the diet will aggravate the effect of an inadequate supply of vitamin D.

Osteomalacia of a severe degree is a rare condition in occidental peoples, and is almost always conditioned by diseases such as idiopathic steatorrhœa or ulcerative colitis; exceptional cases may occur where elderly people live in poverty on inadequate diets and with little exposure to sunshine. It still occurs endemically in wide areas in India and China, but even here its onset is usually conditioned by the extra demands for calcium and vitamin D occasioned by pregnancy and lactation in women on a diet already low in calcium, who have been condemned by religious custom to remain indoors constantly or cover themselves completely from the light when they do leave their homes.

The combined effect of a low calcium intake and a deficiency of vitamin D is to produce a negative calcium balance, and th constant drain of calcium from the body leads to demineralizatio of the bones, and if severe enough, a fall in the level of the blood calcium. The demineralization of the bones leads to the replacement of bony substance by osteoid tissue, and the bones become pliable. The spine, pelvis and extremities are principally affected.

The first symptom in osteomalacia is pain of a deep-seated aching character, usually in the bones of the lumbo-sacral and iliac regions, and often worst at night. As the condition progresses, the pain spreads to other areas and tenderness on pressure of the bones, often very marked, can be elicited.

Bony deformities next develop, and these may occur with surprising rapidity. They are most common in the bones of the trunk – the spine, pelvis and thoracic cage, but those of the lower limb are often involved. The pelvic deformities may be gross, and make parturition difficult or impossible. Due to the osteoporosis, fractures from trivial causes are common. These heal slowly or not at all.

The blood calcium is low, and tetany is therefore a frequent complication of osteomalacia.

Vitamin D and Dental Decay

The view that lack of calcium and vitamin D in the diet leads to defective architecture of the teeth and so predisposes to their decay was discussed in Chapter I (see p. 11). This theory arose as a result of animal experiments in which it was found that rachitic puppies had defective enamel and hypoplastic dentine, and the observation that in rachitic children the teeth are slow in erupting and are subject to early decay. Several investigations in children have afforded evidence that the addition of vitamin D to the diet lowers (though it does not completely eliminate) the incidence of caries, and tends to arrest caries that has already developed.

Many arguments have, however, been adduced against the theory that vitamin D deficiency causes hypoplasia of the teeth and predisposes to dental caries. Thus, some workers have found that administration of vitamin D had no effect on the teeth of children even when given for over a year, and others have pointed to the lack of correlation between rickets and dental caries – children with extensive caries often showing no signs of active or healed rickets and some children with severe rickets having no caries. This last observation has received strong support from an investigation by Wilson and Widdowson (1942) of children in the Kangra valley in India which is notorious for the high incidence of rickets and osteomalacia among its inhabitants; in every case of rickets among the children examined the teeth were nearly perfect.

In experimental rickets in animals, the teeth show the same lack of calcification as the bones, and Irving (1943) has shown that this defect is rapidly corrected by vitamin D administration. It is much less certain, however, whether vitamin D deficiency affects the enamel, the site where defective structure might be expected to predispose to dental caries, and in face of the contradictory findings described above, the verdict as to the causal relationship between vitamin D deficiency and dental caries must at present be the Scots one of 'not proven'. It is not out of place here to refer once more to the importance of vitamin A for the proper development of the enamel of the teeth, and the effect of vitamin C deficiency on the structure of the dentine, although it must be pointed out that, as in the case of vitamin D, opinion is still divided as to whether vitamin C deficiency is a predisposing factor in the development of dental caries.

Requirements and Dosage

The International Unit of vitamin D corresponds in antirachitic activity to 1 milligram of the international standard solution, which is equal to 0·025 microgram of crystalline calciferol (vitamin D_2).

As we have seen, vitamin D is obtained by higher animals in two ways – by the action of the ultra-violet rays of the sun on a sterol in or on the skin, and by the ingestion of food containing the vitamin; it is therefore impossible to assess the exact requirement. Only a few of the natural foods eaten by man contain vitamin D, and then only in small amounts. It would therefore seem that the natural way of obtaining the vitamin is by the action of ultra-violet irradiation on the body, but in temperate climates, under modern conditions of clothing, accommodation and the interfering action of smoke in the atmosphere on the passage of the ultra-violet rays of the sun, it is certain that enough vitamin D is not obtained from this source, particularly in periods of increased requirement, and that a dietetic source of vitamin D is required in addition.

The intimate relationship between vitamin D and normal bon development explains the special need for this vitamin durin growth, and especially in infancy, when there is less exposur to sunlight than in older children.

Full-term breast-fed infants seldom develop rickets, but premature and artificially-fed infants do unless they receive a supplement of vitamin D in their diet, and the question therefore arises as to how big this supplement should be. Amounts as small as 100 International Units per day will protect against rickets, and a breast-fed infant receives less than this in the maternal milk during the first few months, even if the vitamin D content of the milk is raised to the maximum attainable level of some 20 International Units per pint by the administration of vitamin D to the mother. Maximum growth is, however, not secured by amounts of vitamin D that will just prevent rickets, and it is generally agreed that breast-fed infants should be given a vitamin D supplement containing about 400 International Units of the vitamin from the end of the first month, while premature and artificially-fed infants should receive about twice as much. The doses of vitamin D recommended above may be administered in the form of cod-liver oil, of which one teaspoonful contains about 400 International Units. The oil can be given in a teaspoon or dropped on the tongue – it should not be mixed with the milk in the feeding bottle, as it tends to stick to the sides of the bottle so that the infant does not receive the intended dose. If for any reason, cod-liver oil is not well tolerated, the appropriate dose of some of the more highly concentrated vitamin D preparations may be given instead.

Susceptibility to rickets seems to decrease with increasing age, but there is evidence that a certain proportion of children show a poor retention of the calcium and phosphorus of the diet, and that this retention can be improved by vitamin D administration. As it is impossible to detect which children show this poor retention of the bone minerals without elaborate investigation, it is highly desirable that *all* children should be protected by a daily supplement of 400 to 800 International Units for several years, certainly up to the age of 12. Most older children can probably satisfy their requirements from the usual amount of exposure to sunlight, but the occasional occurrence of late rickets during the period of rapid growth at puberty and in adolescence indicates that this cannot be relied on in every case, and ideally a supplement should be continued throughout adolescence.

The question whether or not the normal adult receives sufficient vitamin D from exposure to sunlight and from the small amounts present in a few foodstuffs is not yet settled. Most of the published work indicates that adequate absorption and utilization of calcium and phosphorus occurs when the intake of these minerals in the diet is optimal, irrespective of whether or not a vitamin D supplement is given, and that the administration of vitamin D will not compensate for an inadequate intake of calcium and phosphorus. The relative frequency with which senile osteoporosis occurs and the fact that elderly subjects suffering from this complaint can retain calcium and phosphorus readily if the dietary intake is adequate and vitamin D is given has led to the inference that senile osteoporosis is the expression of a long-continued, mild vitamin D deficiency. But the diet of anything up to 30 per cent of the population of this country is, or certainly was in pre-war days, deficient in calcium (see Table 5), and the experimental work mentioned above would support the view that senile osteoporosis can probably be attributed to a long-continued dietary deficiency of calcium rather than of vitamin D.

It is generally agreed, however, that women should be given a supplement of vitamin D during pregnancy and lactation, both to secure the best possible retention of calcium and to increase the vitamin D content of the maternal milk to the highest attainable level. Administration of vitamin D will not compensate for a defective intake of calcium and phosphorus, but when there are adequate amounts of these minerals in the diet, the taking of additional vitamin D appears to increase their retention. The average vitamin D content of human milk from women in towns is said to be about 17 International Units per pint; administration of vitamin D raises this to (but not above) about 20 International Units per pint, which suggests that the latter is the optima level and that lactating women in towns do not obtain enoug vitamin D from sunlight and from their diet to achieve thi level. A daily intake of 400 to 800 International Units o vitamin D is the recommended supplement in pregnancy an lactation.

In the treatment of active rickets, larger doses are require than those sufficient for protection. Individual needs vary greatl and the correct dose is of course that which cures; abou 1,200 International Units per day is sufficient for most cases o average severity. About three weeks are usually required t assess the effectiveness of a given dose. If this is adequate, th concentration of inorganic phosphorus in the serum should begi to rise in about ten days, and in three weeks the serum phosph tase should have begun to fall and characteristic signs of heali should appear in X-ray photographs of the affected bones. Th mere appearance of these signs is not evidence of complete cure, however, and administration of the effective dose should be continued as long as the phosphatase activity of the serum is higher than normal, since the serum phosphatase remains elevated until cure is obtained. When the rickets has been controlled, the dose can gradually be reduced, but a protective dose must, of course, be continued.

Certain cases of rickets, especially late rickets, fail to respond to the usual doses of vitamin D, and require much higher doses, 50,000 to 100,000 International Units, and in rare cases as much as 1,000,000 International Units, before an effect is obtained. While there is considerable individual variation in the amount of vitamin D that will cause toxic symptoms, doses of 30,000 International Units or over in infants and young children or 200,000 International Units or over in adults may be toxic, and if such doses are being prescribed, a careful watch must be kept for evidence of toxicity.

Symptoms of toxicity appear only some weeks after initiation of treatment with excessive dosage of vitamin D. They consist of anorexia, nausea, headache, diarrhœa, and polyuria. The serum calcium and phosphorus are usually, but not invariably, elevated, and toxic symptoms have been reported in the presence of normal blood levels for these two minerals. In general, however, a serum calcium level of 12 milligrams per 100 c.c. or over should always be considered as a danger signal. If the serum calcium is below this level and there is no radiological proof of healing, it is safe to increase the dose.

Natural Sources

Vitamin D occurs in significant amounts in only a few foods, and man and other animals are therefore mainly dependent for their supply of vitamin D on the action of the ultra-violet rays of the sun. The common foods containing vitamin D are eggs, milk, butter and the body oils of certain fish like salmon, herrings and sardines. All margarine manufactured for table use in Great Britain is now compulsorily fortified with vitamin D (see page 173).

Food sources cannot therefore be relied upon to supplement satisfactorily the vitamin D intake derived from the action of sunlight, and as has been described above, special attempts must be made to combat the deficiency at least during periods of increased requirements. The use of cod-liver oil in this respect is traditional, while modern achievement in the manufacture of purified vitamin preparations has made a number of concentrated sources of vitamin D available.

Official and Proprietary Preparations

A list of preparations containing vitamins A and D is given on p. 99. The following preparations are of vitamin D only:

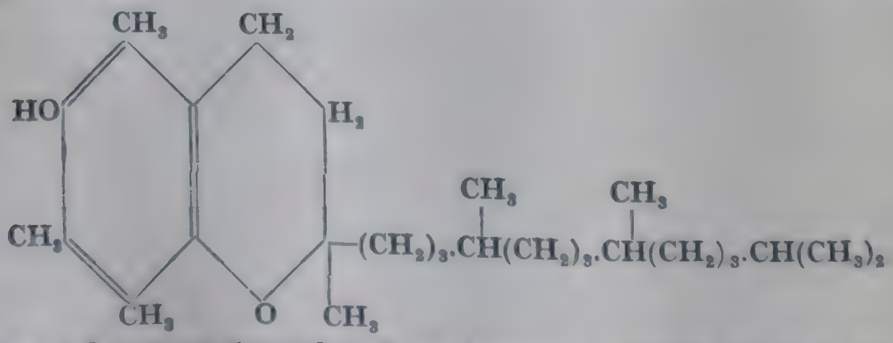
	International Units of Vitamin D
Liquor Calciferolis (B.P. Addendum 1936)	3,000 per gram
Liquor Vitamini D Concentratus (B.P. Addendum 1940)	10,000 per gram
Davitamon D (Organon)	5,000 per c.c.
Davitamon D Forte (Organon)	12,500 per c.c. 12,500 per ampoule
Davitamon D Superforte (Organon)	300,000 per c.c. 300,000 per ampoule
Ostelin (Glaxo)	5,000 per c.c. 500 per tablet
Radiostol (B.D.H.)	3,000, 10,000, or 20,000 per gran 3,000 per pellet
Viosterol (Allen & Hanbury)	15,000 per gram
Vitamin D 'Pabyrn' (Paines & Byrne)	2,000 per tablet 15,000 in 1 c.c. per ampoule

Vitamin E

Chemical Nature

Vitamin E is the generic name given to a group of naturall occurring substances in plant foods, the tocopherols, whic possess in varying degrees the biological activity attributed t this vitamin. The three most potent of the tocopherols in preven ing sterility in the rat are alpha-, beta-, and gamma-tocophero the first having about twice the biological activity of the oth two.*

* Alpha-tocopherol is a complex alcohol with the following stru tural formula:



Beta- and gamma-tocopherol are isomers, possessing one less met group than alpha-tocopherol in the aromatic nucleus.

Alpha-tocopherol is a yellow viscous oil readily soluble in all the lipoid solvents. It is a very stable compound, being resistant to heat up to about 200° C., and to the action of strong acids and alkalis. Prolonged irradiation with ultra-violet light leads to some destruction of vitamin E, but the most potent source of loss is the extraordinary sensitivity of vitamin E to oxidation by slight and obscure oxidative changes occurring in rancid fat.

Crystalline derivatives of the tocopherols have been made, and alpha-, beta-, and gamma-tocopherol have all been synthesized in the laboratory, alpha-tocopherol being now produced on a commercial scale.

Physiological Function and Effects of Deficiency

There is no evidence that animals can synthesize vitamin E and they are probably dependent on plant tissues for the vitamin.

Little is known as to how vitamin E is absorbed, but the presence of bile in the intestine appears to be necessary, and by analogy with the absorption of the other fat-soluble vitamins, fat in the diet should aid absorption.

The young at birth have at best low stores of vitamin E, but adult animals can apparently store vitamin E for considerable periods. The site of these body stores is still a matter of controversy, but the liver contains considerable amounts when the intake of the vitamin is high, and it is found also in the muscles and in the body fat.

Vitamin E is rapidly destroyed in the body when large amounts are taken, while a certain proportion escapes absorption and appears in the fæces. Experimental work suggests that much of the destruction of vitamin E in the body occurs in active, but not in resting muscles.

The fundamental action of vitamin E is still unknown. From its chemical structure, it has been argued that it may take part in oxidation-reduction reactions in the body. Another suggestion, based on changes in the pituitary gland in animals fed on a vitamin E-deficient diet, is that the vitamin is necessary for the normal functioning of the anterior lobe of the pituitary, and that the recognized effects of lack of vitamin E in the diet are secondary to a pituitary deficiency. Most workers consider, however, that the pituitary changes are secondary to the other effects of vitamin E deficiency. A third explanation advanced for the action of vitamin E is that it is necessary either for the synthesis or maintenance of the chromatin of the cell nucleus.

There is great variation in the response of different species of animal to a deficiency of vitamin E, but the major effects are on the reproductive system or the voluntary muscles.

(*a*) *Effects on the Reproductive System*

In the rat, the animal in which the effect of vitamin E on the reproductive system was first discovered and has been chiefly studied, both the female and the male are affected, but the results are so different that they are best described separately.

In the female reared on a vitamin E-free diet, conception occurs normally, but fœtal development lags, and death of the fœtus and its resorption occurs. This failure of reproduction can, however, be cured promptly for subsequent pregnancies by the addition of vitamin E to the diet.

In the male, lack of vitamin E in the diet produces a testicular degeneration affecting first the most mature cells, the sperms, but later involving the germinal epithelium. In contradistinction to the effect of the vitamin in the female, complete degeneration of the testes cannot be averted by the administration of vitamin E

It has been suggested that the reason for the difference in the curative effects of vitamin E in the male and female is that in the male the lesion due to vitamin E deficiency is in the animal's own tissues, whereas in the female the effect of the deficiency is on the fœtus. Degeneration of the smooth muscle of the uterus and brown discoloration due to the presence of small yellow granules in the muscle cells are, however, found in vitamin E-deficient animals and may play a part in the failure of pregnancy

(*b*) *Effects on the Voluntary Muscles*

In a number of species of test animals, vitamin E-deficiency lead to a primary degeneration or dystrophy of the voluntary muscle in which the nervous system plays no part. In fact, the dystrophy can be prevented in individual muscles by destroying tonus either by section of the nerve or tendon of the muscles.

The histological picture – progressive degeneration of individua muscle fibres and their replacement by fat and fibrous tissue closely resembles that of progressive muscular dystrophy in man Chemically there is a reduction in the creatine content of th muscles and an increase in the creatine excretion in the urine The latter is the first evidence of the dystrophy and appear before any clinical signs can be detected.

Administration of vitamin E to animals suffering from muscula dystrophy brought about by lack of the vitamin produces rapid relief. The urinary creatine may fall to normal within two days, and there is a rapid recovery of strength, so that animals hardly able to stand may show no signs of dystrophy by the end of a week, the cure being permanent and complete.

In the rat, muscular dystrophy is accompanied by degenerative changes in the nervous system, affecting the lumbar cord, where there is degeneration of the proximal parts of the posterior roots, the posterior columns, and the anterior horn cells. Vitamin E administration is apparently ineffective in reversing these changes, except possibly in the earliest stages.

Vitamin E deficiency also leads to impaired growth in test animals, which is cured by administration of the vitamin.

Effects of Vitamin E Deficiency in Man

While the effects in animals are well established, it must be admitted that there is no conclusive evidence that a deficiency of this vitamin occurs in man, and the attempts to apply clinically the knowledge acquired of its effects in animals have so far resulted mainly in controversy.

Vitamin E therapy has been used in the treatment of habitual abortion in women, on the grounds that the condition might represent a vitamin E deficiency. Favourable results have been reported by a number of workers, percentages of cure as high as 70 to 80 being claimed. These reports have been criticized by other workers who have stated that equally good results are obtained with endocrine preparations and general measures. It has been claimed, however, that statistical analysis of the most carefully-controlled observations among the published clinical data does indicate an apparently significant relationship between the administration of vitamin E and the cure of habitual or threatened abortion. It therefore seems justifiable to try the effect of vitamin E in the treatment of such cases which are not associated with local conditions such as infection or anatomical derangements, or with obvious endocrine disturbances.

Claims have been made that histological changes similar to hose associated with vitamin E deficiency in animals have been ound in human testes. The lack of curative effect of vitamin E n the testicular degeneration of vitamin E-deficient rats suggests hat, even if similar lesions do occur in man, it is extremely unlikely that they would be relieved by vitamin E therapy.

The similarity between the nutritional muscular dystrophy that develops in animals fed on a vitamin E-free diet and th clinical disease as it occurs in man has led several workers durin the last five years to investigate the effect of vitamin E therap in the human disease. The early reports, both in England an America, of the treatment of muscular dystrophies were encoura ing, but since then, reports denying the value of vitamin E hav been published. In a recent review of the subject, Bicknell an Prescott (1942) defend the favourable reports and state that th negative results may be due to the use of excessive doses alpha-tocopherol unbalanced by other vitamins and to its bein injected instead of given by mouth. They state that wheat ger in small amounts gives better results than synthetic vitamin and attribute this to the presence of other essential nutrien such as vitamins of the B complex in the wheat germ. On negative evidence has so far been obtained by workers who hav investigated the question of whether vitamin E administratio reduces the creatinuria of muscular dystrophy in man.

Requirements and Dosage

The International Unit of vitamin E is the amount which, whe administered orally, has the same potency as 1 milligram synthetic alpha-tocopheryl acetate in preventing resorptio gestation in rats deprived of vitamin E. Nothing is known of th human requirements of vitamin E.

In the treatment of conditions considered to be due to vitamin deficiency, there are three forms in which the vitamin may be give

(*a*) Synthetic alpha-tocopherol. The only advantages of th synthetic vitamin rest in its ready availability and the la of limitation to the size of the dose that can be given. T latter is of doubtful benefit, as there is no evidence th large doses ever need be given.

(*b*) Wheat germ oil. This contains the other tocopherols wheat germ as well as alpha-tocopherol. Like the synthet vitamin, however, wheat germ oil contains none of t other essential factors of wheat germ; moreover, it loses potency on keeping.

(*c*) Whole wheat germ. This is preferable to both the synthe vitamin and wheat germ oil, in that it contains the otl essential nutrient substances of wheat germ, and becau the antoxidants of the wheat germ protect vitamin E fro destruction by rancid fats during digestion. Reliable p parations of wheat germ keep well without becoming ranci

In the treatment of habitual abortion, various workers have used different preparations of vitamin E, such as 4 to 8 c.c. of wheat germ oil or 3 milligrams of synthetic vitamin E daily.

The doses recommended by the advocates of vitamin E therapy in muscular dystrophy are of the order of 6 milligrams daily of alpha-tocopherol in the form of wheat germ oil or a concentrate, or preferably about 1 oz. of whole wheat germ daily taken with milk like a breakfast cereal.

The administration of alpha-tocopherol by injection is not recommended – it has been stated that injection of a large dose of alpha-tocopherol is followed by a fall in the level of vitamin E in the blood, whereas oral administration causes a rise.

Natural Sources

The richest sources of vitamin E are green leafy vegetables, particularly lettuce, and the embryos of certain seeds (such as barley, maize, rice, oats, cotton seed, peanut, soya bean, and especially wheat), in which the vitamin occurs in the unsaponifiable fraction of the oil. Small amounts occur in fat and muscle tissue, milk, cheese, butter and eggs, but accurate data as to the amounts of vitamin E in foods, and the adequacy or otherwise of human diets in this vitamin are still lacking. It can, however, be stated that wholemeal flour constitutes one of the two most important dietary sources of vitamin E, the other being green leafy vegetables. These two foods formed anything but prominent parts of the diet of the poorer section of the population of England in pre-war days, and it is a matter of speculation as to whether the small amount of vitamin E that must have been present in the diet was adequate. In contrast to the position in England, the diet of even the poorest people in France has always contained a high proportion of vegetables, and according to Drummond and Wilbraham (1939) habitual abortion is almost unknown in that country.

Proprietary Preparations

Davitamon E (Organon)	Capsules containing 0·5 c.c. wheat germ oil
Ephynal (Roche) .	Tablets containing 3 mg. tocopheryl acetate
Fertilol (Vitamins Ltd.)	Capsules containing 5 minims of wheat germ oil
Phytoferol (B.D.H.) .	Capsules equivalent to 3 mg. alpha-tocopherol

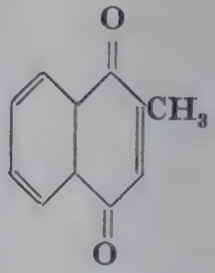
Vitamin E (Allen & Hanbury) . . .	Capsules equivalent to 3 mg. alph tocopherol
Vitamin E (Burroughs Wellcome) . .	Tabloids equivalent to 8 mg. alph tocopherol
Viteolin (Glaxo) . .	Capsules equivalent to 6 mg. alph tocopherol

Vitamin K

Vitamin K is the term applied to a group of naturally-occurri substances recently detected in plant and animal foods, in tl absence of which a hæmorrhagic tendency develops, due to a fall the level of prothrombin in the blood.

Chemical Nature

Concentrates of vitamin K were first obtained by extracting alfal (lucerne) or fish meal with fat solvents, and later two chemical different compounds were isolated from these substances, that fro alfalfa being termed vitamin K_1 and that from fish meal K These two substances both contain a derivative of naphthoquinor 2-methyl, 1 : 4-naphthoquinone, whose structural formula is:



They possess different side-chains attached to this nucleus, th of vitamin K_1 being phytol. Many substances related to vitamin and having similar biological activity have been synthesized; m of these are derivatives of 1 : 4-naphthoquinone or 1 : 4-naphtl hydroquinone. The derivative forming part of the molecule of bo naturally-occurring forms of vitamin K, 2-methyl, 1 : 4-naphtl quinone, has, molecule for molecule, a biological activity equal that of the natural vitamin, and being a smaller molecule, it almost three times as active weight for weight. It has been includ in the British Pharmaceutical Codex under the official name Menaphthone; in the United States it is also an official preparati under the name of Menadione.

The naturally-occurring forms of vitamin K are fat-solul inactivated by light in a few hours, and destroyed by strong ac and alkalis, and by oxidizing agents. The synthetic compou Menaphthone is likewise fat-soluble and inactivated by light. I only very slightly soluble in water and hence cannot be given int venously, but closely-related compounds that are soluble in water have been prepared and can be administered by intravenous injection.

No toxic effects in man have been reported from the administration in reasonable therapeutic amounts of either the natural vitamin or any of its synthetic analogues, although in animals excessive doses are toxic and even lethal, producing respiratory depression and acute vascular congestion.

Physiological Functions

So far as is known, vitamin K has only one function in the body, namely, to maintain a normal level of prothrombin, in the blood. It is not known whether vitamin K is a constituent of prothrombin or whether it acts by stimulating the liver to produce prothrombin.

The clotting of blood occurs in two stages. When blood is shed, calcium and prothrombin, both present in normal blood, interact with thromboplastin (thrombokinase), formed only when tissues are injured or blood platelets undergo disintegration, to produce thrombin. The latter substance then converts the soluble plasma protein, fibrinogen, into the insoluble fibrin. Vitamin K is important only in one part of this mechanism, namely, the maintenance of an adequate supply of prothrombin in the blood, and is therefore ineffective in the treatment of hæmorrhage in normal persons or where a hæmorrhagic tendency is due to causes other than a low blood prothrombin level. The term 'antihæmorrhagic vitamin' for vitamin K is thus too sweeping. It must be remembered, too, that vitamin K is not the only factor concerned in the production of prothrombin. The liver is the site of prothrombin formation, and severe liver damage is one other factor at least that may cause a fall in the blood prothrombin level and bleeding irrespective of the amount of vitamin K available.

Bile is essential for the proper absorption of vitamin K, which is stored in the liver after absorption. This storage is evidently very limited, however, as severe hypoprothrombinæmia can occur very rapidly after the onset of conditions interfering with the availability of vitamin K to the body.

Vitamin K is found in the fæces. It has been shown that intestinal organisms, in particular *B. coli*, can synthesize vitamin K, but it is not yet known to what extent this can meet the body's needs for vitamin K and render it independent of a dietary source of the vitamin. Recent reports of the occurrence of dietary vitamin K deficiency in man suggest that a preformed dietary supply is necessary in addition to whatever amount may be derived from bacterial synthesis in the intestine.

Effects of Deficiency

A fall in the level of prothrombin in the blood due to a deficiency of vitamin K may occur in either of two ways:

(i) *Ingestion of a Diet deficient in Vitamin K*

In some species of experimental animal, the administration of a vitamin K-deficient diet causes a fall in the blood prothrombin level, indicating that bacterial synthesis of the vitamin in the intestine is insufficient to meet normal requirements.

Whether a similar condition can occur in man is still a matter of some uncertainty, but more than one worker has reported the finding of a lowered blood prothrombin level in patients who were not suffering from any disease of the liver, bile ducts, or intestines, and its prompt restoration to normal on administration of vitamin K. Most of these patients had consumed a generally inadequate diet, and showed evidence of other nutritional deficiency diseases.

Hypoprothrombinœmia of the newborn, in which during the first few days of life there is a deficiency of prothrombin in the blood, may perhaps be considered under this heading, although the cause of this normal physiological deficiency is still controversial. In all infants there is a natural drop in the prothrombin level of the blood during the first 2 or 3 days after birth. It is generally attributed to the supply of vitamin K from maternal sources being inadequate to carry the infant over the first few days before establishment of the flora of the intestine and the commencement of bacterial synthesis of vitamin K therein. The fall is usually not of sufficient magnitude to impair the clotting power of the blood to such an extent that hæmorrhage occurs, but in some cases, the drop in the blood prothrombin is exaggerated to a pathological extent and hæmorrhages result. The condition at this stage is characterized by multiple hæmorrhages in any site but especially from mucous surfaces, and bleeding may occur into the skin, or from the mouth, nose, stomach, intestines, kidneys, lungs and the membranes of the brain. These hæmorrhages are apparently spontaneous; in reality, however, they are initiated by trauma, although this may be very slight and unnoticed, the persistence of the bleeding being due to the impaired clotting power of the blood resulting from the fall in its prothrombin level. The hæmorrhages rarely begin before the second or third day after birth, and if untreated the condition ends in death or in spontaneous recovery, usually by about the beginning of the second week.

The symptoms are those of hæmorrhage, which is usually visible in the skin or mucous membranes, but often may be marked only by hæmatemesis, melæna, hæmaturia, or cerebral hæmorrhage. Fever may or may not be present, and the infant frequently becomes prostrated and develops diarrhœa. Death results from blood loss and general debility. It has been suggested that many of the intracranial hæmorrhages which sometimes follow a protracted labour and lead to permanent paralysis or mental impairment, if not immediately fatal, are an expression of a vitamin K deficiency, in that the bleeding due to the birth trauma would have been slight and of no significance had it not been for the diminished prothrombin level and impaired clotting power of the blood.

The defective mechanism in hæmorrhagic disease of the newborn is not directly a matter of inadequate supply from the mother, since there is no correlation between the prothrombin level of the infant's blood and the intake of naturally-occurring vitamin K in the maternal diet. As described under treatment, however, the administration of synthetic products like menaphthone to the mother just before labour will raise the prothrombin level of the infant's blood up to or nearly up to a normal level, suggesting that hypoprothrombinæmia of the newborn may be due to difficulty in the transfer of vitamin K across the placenta towards the end of pregnancy.

(ii) *Inadequate Intestinal Absorption*

As mentioned already, the presence of bile salts is essential for the absorption of vitamin K. Hence diseases of the liver or biliary passages which lead to defective secretion of bile into the intestine impair the absorption of vitamin K, and form the most important group of disorders leading to a conditioned deficiency of vitamin K in adults, and a fall in the level of the blood prothrombin to hæmorrhagic levels. Hypoprothrombinæmia is now recognized as the cause of the hæmorrhagic tendency in jaundice, which not only may lead to purpuric and other hæmorrhagic manifestations, but also constitutes a dangerous and always threatening complication of surgical procedures on jaundiced patients.

Other intestinal disorders, however, such as steatorrhœa, or ulcerative colitis and short-circuiting operations on the alimentary tract which do not specifically affect fat absorption, may lead to a deficient absorption of vitamin K and a resultant fall in the level of blood prothrombin. This is the explanation of many of the otherwise obscure intestinal hæmorrhages that occur in these conditions.

Injury to the Liver

The liver plays an active part in the formation of prothrombin, and any severe injury to this organ results in a deficiency of prothrombin. In contrast to the other two causes of this deficiency, however, administration of vitamin K or any of its synthetic analogues will not restore the blood prothrombin to normal, since the defective prothrombin production is not due to an inadequate supply of vitamin K but to damage of the hepatic cells in which it is manufactured. Hypoprothrombinæmia due to liver damage is therefore not due to a vitamin K deficiency, conditioned or absolute, and has been included in this discussion in order to emphasize that hypoprothrombinæmia is not a specific sign of vitamin K deficiency.

Diagnosis of Hypoprothrombinœmia and Vitamin K Deficiency

The hypoprothrombinæmia due to vitamin K deficiency is readily corrected, both before and after it has reached the stage of frank hæmorrhage, by the administration of vitamin K or one of its synthetic analogues. Hæmorrhage is, however, a late manifestation of vitamin K deficiency, and diagnosis before this stage is reached is highly desirable, particularly in patients who are to be subjected to any form of surgical procedure.

The dietary history is unlikely to be of much help in diagnosis, since vitamin K deficiency in adults is almost always a conditioned one, due to diseases like those mentioned already which prevent absorption of the vitamin even in the presence of an adequate intake. The possibility of a complicating vitamin K deficiency must therefore always be considered in such diseases.

The deficiency is detected by estimation not of the vitamin K content, but of the prothrombin content of the blood. In the absence of other conditions affecting the blood prothrombin. *e.g.* severe liver damage, the finding of a hypoprothrombinæmia affords strong presumptive evidence of vitamin K deficiency. Several methods of determining the prothrombin content of the blood have been devised, but the one most widely employed is the method of Quick, in which the clotting time is determined for oxalated plasma to which an excess of thromboplastin and a fixed amount of calcium have been added. By this method, a normal concentration of prothrombin results in a clotting time of 15 seconds or less.

Requirements and Dosage

The normal daily requirements in man are unknown. The uncertainty whether vitamin K deficiency ever occurs in the absence of some disease interfering with its absorption suggests that the normal requirements are easily met from the liberal supply contained in any ordinary diet together with the vitamin K formed in the intestine by bacterial action.

Prevention and cure of vitamin K deficiency need only be considered, therefore, in the case of two special groups: the newborn infant and patients suffering from any of the diseases likely to interfere with absorption of vitamin K.

(*a*) *Newborn infants* can be protected from hypoprothrombinæmia by administration of vitamin K either to the mother before delivery or to the infant after birth. There is evidence that the former procedure is more effective in that it raises the blood prothrombin to a greater extent. While some workers advocate that the mother should take 1 milligram of menaphthone or its equivalent by mouth daily for the last month before delivery, others state that such treatment for three to four days prior to delivery or a single dose of 2 milligrams of menaphthone by mouth at any time from one to forty-eight hours before delivery is effective in preventing hæmorrhagic disease of the newborn infant. If the mother is not treated until labour has begun, reliance should not be placed on oral therapy owing to the delay in absorption, and she should be given an intravenous injection containing 4 milligrams of a water-soluble vitamin K derivative, or an intramuscular injection of 6 milligrams menaphthone in oil. Where it is decided to treat the infant directly 1 milligram daily in divided doses for the first two or three days after birth will tide the infant over the first critical week when the blood prothrombin falls and a hæmorrhagic tendency may develop. The vitamin solution can be placed in drops on the back of the infant's tongue.

There is no correlation between the dietary intake of vitamin K or the level of prothrombin in the maternal blood and that in the blood of the infant after delivery, and therefore no means of predicting which infants are likely to suffer from an aggravated degree of hypoprothrombinæmia. For this reason, most workers advocate that vitamin K in some form should be administered to every mother before delivery. The cheapness, availability, and lack of toxic effect of the vitamin make this no extravagant suggestion. If adopted it would eliminate the danger of hæmorrhagic disease of the newborn and possibly reduce markedly the incidence of cerebral hæmorrhage following birth injury. Where the infant is first seen with actual hæmorrhage or with a blood prothrombin reduced to bleeding levels, prompt treatment with vitamin K is imperative. In most cases, the same dosage as that for protection – 1 milligram daily in divided doses for about three days – is adequate; or 1 to 2 milligrams may be given parenterally and repeated daily until the hypoprothrombinæmia is corrected.

(*b*) *Patients suffering from vitamin K deficiency induced by diseases of the liver, bile-duct or alimentary tract* may be treated orally provided that the vitamin K preparation is administered in conjunction with bile salts, particularly where jaundice is present. Theoretically bile salts alone, by improving absorption, should be adequate to relieve vitamin K deficiency and in some cases have been shown to be so. But in practice, where rapid correction of the hypoprothrombinæmia is desirable, vitamin K should be given as well.

A dose of 1 to 2 milligrams daily of menaphthone or other vitamin K analogue plus 10 to 15 grains of bile salts is usually adequate. If this treatment does not raise the level of the blood prothrombin, the doses should be increased up to 5 milligrams daily, or injections of 1 to 2 milligrams of a vitamin K preparation in oil may be given intramuscularly or a water-soluble derivative intravenously.

Since the fall in blood prothrombin to bleeding levels caused by vitamin K deficiency may develop rapidly, it is probably wisest to administer protective treatment for some days, both before and after operation, to all patients suffering from a disease likely to interfere with vitamin K absorption in whom surgical intervention is necessary, irrespective of the initial level of the blood prothrombin.

In patients with jaundice or digestive diseases in whom the blood prothrombin is so low that hæmorrhages have developed, vitamin K therapy must be started at once. An initial dose of 1 to 3 milligrams of menaphthone or its equivalent should be given, followed by a similar dose in 12 hours and thereafter daily. If this is not sufficient to raise the prothrombin level, the doses should be increased up to 5 milligrams daily. Each dose of vitamin K should be accompanied by one of bile salts in doses from 10 to 30 grains, depending on the size of the accompanying dose of vitamin K. Where administration by mouth is difficult, parenteral injections of 1 to 2 milligrams of a vitamin K analogue should be given at 6 to 12-hour intervals.

Where the liver has suffered severe damage, there supervenes hypoprothrombinæmia that does not respond to vitamin K therapy. There is some evidence that abundant vitamin K may enable a less severely damaged liver to maintain a normal blood prothrombin level when otherwise it could not have done so. A trial of vitamin K therapy should therefore be made even in hypoprothrombinæmia thought to be largely due to liver damage; though for the treatment of this condition chief reliance should be placed on blood transfusion.

Natural Sources

Vitamin K is widely distributed in Nature, among the chief sources being green leaves of different kinds. Alfalfa and spinach are particularly rich in the vitamin, but cabbage, cauliflower, carrot tops, and kale are all good sources. Seeds, fruits and roots in general contain much less of the vitamin than green leaves. It occurs in soya bean oil, egg yolk, and putrefied fish meal; but although fat-soluble and obtainable from fish meal, it is nevertheless not found in fish-liver oils.

Proprietary Preparations

Davitamon K (Organon)	Acetomenaphthone, 10 mg. per tablet, or ampoules containing 10 mg. in 1 c.c.
Kappaxan (Bayer) .	Acetomenaphthone, 10 mg. per tablet, or menaphthone, 5 mg. per ampoule
Kayvisyn (Parke, Davis & Co.) . . .	1 mg. of 4-amino-2-methyl-1-naphthol in 1 c.c.
Menaphthone (Burroughs Wellcome) . .	2 mg. per tablet 5 mg. per ampoule
Prokayvit (B.D.H.) .	5 mg. menaphthone per ampoule 10 mg. acetomenaphthone per tablet 10 mg. acetomenaphthone per ampoule
Synkayvit (Roche) .	10 mg. per tablet or per ampoule of the tetra-sodium salt of 2-methyl-1:4-naphthohydroquinone diphosphate, a water-soluble analogue of vitamin K
Vitamin 'K' Pabyrn .	10 mg. menaphthone per ampoule 10 mg. acetomenaphthone per tablet
Kapilon (Glaxo) . .	Acetomenaphthone, 10 mg. per tablet, or menaphthone, 5 mg. per ampoule.

CHAPTER V

THE MINERALS

EVERY element which takes part in the structure of the cell is essential to life. Therefore, besides the proteins, fats, carbohydrates and vitamins already considered, adequate nutrition demands a supply of the inorganic constituents of the body in their proper proportion. The elements present in the body and definitely concerned with its functioning are:

Metals – calcium, sodium, magnesium, potassium, iron, copper, manganese and cobalt.

Non-metals – sulphur, phosphorus, chlorine and iodine.

The metals are present in the body as the basic part of salts (cations): the non-metals, partly in the acidic part of salts (anions) and partly in organic combination.

Other elements occur in traces in the body, but the part, if any, which they play in the vital processes is not yet known.

The mineral elements of the body have the following important functions:

(1) They form an essential part of the skeletal tissues, over half of the solid material of bone consisting of inorganic matter.

(2) They enter into the composition of many of the organic compounds of the body, for example iron in hæmoglobin, phosphorus in nucleoproteins, and iodine in thyroxine.

(3) They occur in the tissue fluids where they serve to maintain the osmotic pressure and the hydrogen-ion concentration at physiologically healthy levels. The irritability of nervous and muscular tissue is controlled by the balance of certain basic ions in the body fluids. Any increase of sodium or decrease of calcium, for instance, increases the irritability of these tissues.

The Utilization of Minerals. The mineral elements occur in living matter in three forms – in organic combination, as inorganic salts and as dissociated ions. Theoretically, the minerals of the foodstuffs might be absorbed in any of the three forms, and it was at one time considered that minerals in organic combination were utilized by the body more readily than if they were present as inorganic salts. It is now believed that every mineral compound, organic or inorganic, must be split in the intestine and the mineral liberated either as a simple salt or as free ions before it can be absorbed and utilized by the body. The practical importance of this conclusion is that, when it is considered that the body requires an added supply of a mineral, no advantage is gained by prescribing an organic compound of the mineral in question. A simple inorganic salt of the mineral will supply it in a more readily utilizable and usually cheaper form.

The Daily Requirement of Minerals. Many of the minerals required by the body are present in such amounts in any ordinary diet that the body is assured of an adequate supply. There are four, however, in which the diet may fall short of the minimal requirement, namely, calcium, phosphorus, iron and iodine; under special conditions, too, a dietary deficiency of sodium chloride may arise, and the requirements for this salt therefore demand detailed consideration also.

I. Calcium

Calcium is essential to the body for the building of bone and teeth, and the maintenance of a normal blood calcium level is necessary to maintain the optimal irritability of nervous and muscular tissues. It must be remembered that bone is not only a supporting structure. The skeleton is the normal reservoir of calcium in the body, 99 per cent of the total body calcium being present in the bones. The metabolism of bone does not come to a standstill when ossification is complete. Deposition of lime salts in, and absorption from, the trabeculæ of bone occur throughout adult life. The mineral deposit in bone is therefore not fixed, but under changing conditions, physiological as well as pathological, it is as easily removed as it is normally deposited. For example, in the cow, there is a definite negative calcium balance during the milking period, leading to a loss of up to 20 per cent of the body calcium, and a corresponding calcium retention during the dry period, when the depleted body store of calcium is restored. The internal secretion of the parathyroid glands controls the mobilization of calcium from the skeletal stores.

Factors Affecting Absorption and Utilization

Calcium is probably absorbed largely in the upper part of the small intestine where the acidified gastric contents, on passing into the small intestine, keep the reaction somewhat acid. The reaction of the lower part of the small intestine tends to be alkaline and precipitation of calcium occurs there. Factors promoting an acid reaction of the intestinal contents therefore facilitate the absorption of calcium. Moderate amounts of fat in the diet have a beneficial influence on calcium absorption, generall attributed to their action in promoting acidity of the intestina contents, although this explanation has recently been questioned On the other hand, in disturbances of fat metabolism such a idiopathic steatorrhœa, where excessive amounts of split fat ar present in the intestinal contents, the fatty acids form insolubl soaps with calcium and so interfere with its absorption.

For reasons not yet understood, vitamin D increases the acidit in the small intestine, which doubtless explains at least in par its beneficial effect on calcium metabolism in rickets. Furthe important factors affecting the absorption and utilization o calcium are the absolute amounts and relative proportions o calcium and phosphorus in the diet, the state of nutrition a regards calcium, the availability of the dietary calcium, and according to recent reports, the protein content of the diet.

Calcium : Phosphorus Ratio. In general, an increase in calciun intake results in an increased *total* retention, but a fall in th *percentage* retained. The ratio of calcium to phosphorus also ha an important bearing on the metabolism of both these elements Excessive amounts of phosphorus in the diet combine with calcium to form insoluble calcium phosphate and so lead to a increased excretion of calcium in the fæces; similarly an increase intake of calcium leads to a greater fæcal excretion of phosphorus The optimum calcium : phosphorus ratio in the diet is probabl of the same order as the ratio in which these minerals are retained provided that they are furnished in utilizable form in the diet The normal calcium : phosphorus retention ratio in infants i 2 : 1 or over, but it sinks to about 1 : 1 in older children, owin to the relatively greater amount of phosphorus required for sof tissue growth. In adult dietaries, a calcium : phosphorus ratio i the diet of between 1 : 1 and 1 : 2 appears to cover any difference that may occur in the requirements of these minerals unde various physiological conditions. It is of interest to note that, i experimental animals, an excess of phosphorus in the diet is bette tolerated than an excess of calcium.

Previous Nutritive State. There is a considerable amount o suggestive evidence that, both in children and in adults, th nutritive state of the body with respect to calcium is a determinin factor in the amount of dietary calcium retained. Thus, for an particular age group, whose subjects are receiving an equal amoun of calcium, the absorption and retention of dietary calcium appear to be greater in those subjects who have previously had a low than in those who have previously had a generous calcium intake. It is obvious that this variability in calcium retention makes difficult the estimation of dietary needs from balance experiments.

Availability of Dietary Calcium. Calcium in the food is not all equally available to the body. Thus, the calcium of plant foods is generally less well retained than the calcium of milk. The chief factor concerned is the presence of oxalic acid in plant foods, which combines with calcium to form insoluble calcium oxalate and so renders it unavailable to the body. Among the vegetables commonly consumed, those with the highest oxalic acid contents are beets and spinach – spinach containing so much that it not only fails to supply any available calcium itself but also renders unavailable a considerable proportion of that present in other foods ingested at the same time.

Action of Phytic Acid. Another important drain on the dietary calcium is due to the presence of an organic acid, phytic acid (inositol-phosphoric acid), in the pericarp of cereal grains. Phytic acid unites with calcium and magnesium to form the substance known as phytin, which is resistant to destruction in the human alimentary tract, so rendering the minerals present therein unavailable to the body. In two recent reports, McCance and Widdowson (1942) have shown that absorption of calcium, magnesium, and phosphorus is reduced when wholemeal bread is substituted for white bread in the diet, and that the phytic acid of the bran in the wholemeal bread is the factor responsible for this reduction in absorption.

Non-availability of phytin phosphorus to the body has been shown to be the cause of the rachitogenic effect of wholemeal cereals first noted several years ago in experimental animals, and formerly attributed to the presence of an anticalcifying substance in cereals. As explained in Chapter VIII, the undesirable effect of high-extraction flour on the absorption of dietary calcium has been counteracted in Great Britain by the compulsory addition of sufficient calcium carbonate to combine with its phytic acid.

Influence of Protein Intake. It has been shown by McCance and his co-workers (1942) that calcium absorption and retention in normal adults are increased significantly when 100 to 300 grams of protein are added to a basal protein intake of 40 to 75 grams per day. Their explanation is that the amino-acids derived from the digestion of protein combine in the intestine with calcium phosphate and carbonate to form soluble compounds and so facilitate the absorption of calcium. It is also possible that a buffering effect of the products of protein digestion on the reacti of the intestinal contents may be the principal factor involve

Excretion in Urine and Fæces. Calcium is constantly bei excreted both in the urine and in the fæces. Normally, abo 90 per cent of the total calcium excretion is through t bowel. This does not, however, all represent unabsorbed calciu since there is convincing evidence that calcium is excreted in the bowel.

Variations in Utilization. In spite of the various factors tendi to promote the absorption of calcium, man's capacity to utili the calcium in his diet is limited, recent work showing that i average utilization both in children and in adults does not exce 20 to 25 per cent. There appears to be considerable though yet unexplained individual variations from this average. The ra of growth is probably not a determining factor in children, sin there is apparently no correlation between the rate of utilizatio and that of growth in height, nor is there any evidence of individu differences in digestive ability. According to some workers th variations depend on a factor mentioned already – namely, th previous dietary intake of calcium. Where this has been inad quate there will be a greater retention on a liberal calcium intak than where the previous intake has been sufficient to promo maximal calcium storage. It is of interest to note here that th calcium retention found in short balance experiments on childre is almost invariably much higher than the amount theoreticall necessary to maintain optimum skeletal calcification, as estimate from the rate of skeletal growth and the amount of calcium i the skeleton.

Estimates of the dietary calcium requirements will differ ther fore according to whether they are made solely on the basis o calcium balance experiments, or on theoretical calculations base on (*a*) the calcium retention necessary at various ages to maintai normal calcification of the skeleton and (*b*) the average calciu utilization as established in metabolic experiments.

Dietary Requirements

Since calcium is required in large amounts to provide for th growth of the skeleton in children, it is obvious that the requirements during growth will be relatively much greater than i adult life.

Leitch (1936) has calculated the calcium requirements durin growth from the results of metabolism studies and from th retical considerations. On the assumption that 50 per cent of the calcium intake can be retained, she gives the following as the minimum daily requirements:

Age	Calcium requirement in grams
6 months to 2 years	0·8
2 years to 9 years	0·9
9 years to 16 years	1·0 increasing to 2·0 between the ages of 15 and 16, and then gradually decreasing to the adult requirement of 0·55 gram.

A more recent estimate, based likewise on the results of metabolism studies, has been made by Duckworth and Warnock (1942), who calculated the average daily calcium retention at different ages, and from the data of the balance experiments obtained the dietary calcium intakes required to secure these retentions. Their estimates are as follows:

Age	Calcium requirement in milligrams	
	Male	Female
Up to 7 years	700	700
7 years to 8 years	800	800
8 years to 11 years	900	900
11 years to 14 years	1,000	1,000
14 years to 15 years	1,100	900
15 years to 16 years	1,100	800
16 years to 17 years	1,100	700
Adult	700	600
Pregnancy (latter half)		1,500
Lactation		2,000

They point out, however, that these averages do not allow a margin of safety to cover individual variations, such as those occasioned by children reaching puberty at an early age, and suggest that the more liberal allowances recommended by the U.S. National Research Council (see Table 1, p. 2) should therefore be adopted in planning dietaries.

Theoretical estimates, like those of Holmes (1945), are based on the assumption that the child has been well nourished from birth with respect to calcium, and give generally lower values than those quoted above. It is, however, uncertain whether many

children can fulfil the qualification stated, and these est
provide no margin of safety for those children who utilize d
calcium with less than average efficiency. While of theor
interest, therefore, they should not be employed in pla
dietaries without additions to cover the requirements of ch
who have been previously undernourished with respect to ca
or who utilise calcium badly.

The child's diet, apart from milk and milk products, is un
to provide more than about 0·2 gram of calcium. Milk co
approximately 0·68 gram of calcium per pint, so that
calcium intakes recommended in Table 1 (p. 2) are to be se
the diet up to the age of 12 years should contain 1 to 1½ p
milk or its equivalent in cheese (on the basis that 1 oz. of c
equals 7 oz. of liquid milk), while from 12 to 16 (or 20
case of youths), the amount should be 1½ to 2 pints of milk
equivalent in cheese.

The minimal daily calcium intake required to maintain e
rium in an adult is about 0·5 gram. Since calcium is not
readily absorbed, it is customary to allow a 50 per cent ma
safety, so that the daily calcium intake of an adult sho
approximately 0·8 gram. This does not allow for the
demands for calcium during pregnancy and lactation; under
conditions it is essential that the calcium intake be increa
to about 1·5 to 2 grams.

The foodstuffs of the average diet of an adult, other tha
or milk products, are unlikely to contain more than
0·3 gram of calcium. If, therefore, a normal adult takes
¾ pint of milk or its equivalent in cheese daily, his calcium re
ment should be satisfied. To attain the calcium intakes r
mended for pregnancy and lactation, at least 2 pints of milk
be taken daily.

The importance of a rich intake of calcium during lactati
been clearly demonstrated by Leitch (*loc. cit.*). From an a
of a large series of experiments carried out in different co
it was proved that the calcium intake from the mother's m
insufficient in many instances to supply enough of the min
maintain the composition of the growing skeleton at its
level, so that defective calcification of bones must have re
There is abundant evidence that the diet of the mother dete
the yield of milk, and it has been claimed that the calcium c
of the milk can to some extent be modified by feeding.

It was mentioned earlier in this section that the metabo

e does not cease with the completion of ossification. There is a
tant mobilization of lime salts from the bone trabeculæ, and
loss has to be made good from the diet if bones of normal
ngth are to be maintained. Senile osteoporosis was formerly
idered to be the result of lowered functional activity of the
e and degeneration of the organic matrix, with the result that
processes of deposition of lime salts in bone no longer kept
with those of mobilization. The rapid response of cases of
e osteoporosis to treatment with calcium and vitamin D
ws, however, that in old age the bones are still capable of
ing down calcium actively if given the chance, and it is now
d by many authorities that senile osteoporosis is rather the
lt of the intake of a diet deficient in calcium over a large
ber of years. A glance at Table 5 (p. 6) will show that fully
the people of this country do not receive enough calcium in
diets to meet the recommended adequate daily intake of
gram, while the members of the lowest income group, repre-
ing 10 per cent of the population, do not even receive enough
um to meet the minimum requirement for equilibrium,
ly 0·5 gram. It is not surprising that osteoporosis is fre-
ntly found in elderly persons of the poorer classes, who have
umed such inadequate diets for years.

has been claimed by some workers that over long periods
nadequate calcium intake the body develops the ability to
erve this element in an attempt to maintain the normal
iological functions. This view is supported by the studies
ichols and Nimalasuriya (1939) who found comparatively
-developed skeletons in natives of Ceylon who habitually con-
d a low calcium diet. These natives were, however, under-
l as compared with better-fed natives, and much of the
arent adaptation to a low calcium intake may have been due
is stunting of growth which obviously would decrease the
um requirements but can hardly be regarded as physiological.
nalogous instance is the finding that starvation and the con-
nt retardation of growth may actually relieve rickets already
ent. This adaptive mechanism would not prevent the ill-
ts of prolonged calcium deficiency in adults of normal stature,
the above findings do not invalidate the view that senile
porosis may be the expression of a long-continued calcium
iency.

rummond (1938) has pointed out the striking contrast between
nt-day diets as revealed by the dietary surveys carried out

by Sir John Orr and others and the diet of the relatively po Englishman of centuries ago when, thanks to a liberal consumpti of milk and cheese, the daily intake of calcium was often as muc as four to six times that of the poor to-day. For example, seve teenth-century diets of simple folk – peasants or the patients St. Bartholomew's Hospital – provided no less than 2 grams calcium a day and a calcium : phosphorus ratio which was ofte excellent.

Calcium and Vitamin D Deficiencies

The interrelation of calcium and vitamin D is so close that it impossible to discuss the effects of deficiency of one withou consideration of the other.

As we have seen, this vitamin appears to play an importa part in regulating the absorption of calcium from the intestin a higher percentage of the available calcium being absorbed i the presence of an adequate supply of vitamin D and the absolut amount of dietary calcium required being lower than when th intake of the vitamin is low. It is uncertain how far the convers holds and to what extent, if at all, a liberal supply of availabl calcium can overcome a slight inadequacy of vitamin D. It i certainly less likely to do so in children with their enhance demands for skeletal growth than in adults, in whom adequat absorption of calcium occurs when the intake is optimal irrespective of whether or not a dietary source of vitamin D i provided.

Calcium deficiency affects chiefly the bones, the blood serum neuromuscular excitability and the calcium balance of the subject (*i.e.* the balance between the intake and excretion of calcium The calcium balance is always negative in calcium-deficienc states; in other words, more calcium is being excreted than i being retained from the diet. This leads to a progressive reduction in the calcium content of the bones and also a reduction i the actual bony substance, so that the bones are not only mor pliable but are also decreased in substance. Bending and deformit occur as a result. The calcium content of the blood serum i maintained as long as possible by the mobilization of the calciu reserves of the skeleton, but if the deficiency is severe and protracted a point is reached where these reserves are no longe sufficient for the purpose, and the level of calcium in the serum falls. Calcium ions depress neuromuscular excitability, and decreased concentration of calcium ions in the serum leads to a increase in neuromuscular excitability, which finds clinical expression in the various symptoms and signs of latent or manifest tetany.

An inadequate supply of vitamin D is usually associated with a dietary deficiency of calcium in the production of calcium deficiency states of nutritional origin. In children, deficiency of vitamin D leads to the development of rickets even in the presence of an adequate calcium intake, but when a dietary deficiency of calcium is also present, the condition known as spasmophilia (for description see Chapter IV) supervenes as a complication of rickets.

In adults, a deficiency of calcium in the diet leads to the development of osteoporosis or, in severe cases, osteomalacia. It is difficult to say how far a calcium deficiency alone may be responsible for these conditions or whether a coincident deficiency of vitamin D always plays a part in their causation. All that can be said is that the most severe cases of osteomalacia are seen among women in Eastern countries who, as well as consuming a diet inadequate in calcium at least for the added demands of pregnancy and lactation, are also compelled by religious practices either to remain indoors entirely or to cover their bodies completely from light if they do emerge from their homes. The symptoms and signs of osteoporosis and osteomalacia have been described in Chapter IV in the section dealing with vitamin D.

Treatment of Calcium Deficiency States

The treatment of calcium deficiency states of nutritional origin consists in the administration of sufficient calcium together with vitamin D to ensure that the calcium is well utilized. Calcium may be given curatively in the form of its chloride, lactate, or gluconate. It must be remembered that these salts contain different amounts of calcium by weight – about 36 per cent of the chloride, 18 per cent of the lactate, and 10 per cent of the gluconate. Consequently the dose to be administered will vary with the calcium salt prescribed. The usual therapeutic dose of *calcium* in calcium deficiency states is 1 to 2 grams daily in children and 2 to 3 grams in adults. Calcium chloride has an unpleasant taste and causes acidosis if administered over a long period, so that the lactate is commonly prescribed, usually in tablet form, although it can be given in solution in water flavoured with fruit juice.

Oral administration is adequate except in the treatment of manifest tetany where, in addition to the oral treatment describ above, intravenous injections of 10 c.c. of a 10 per cent soluti of calcium gluconate should be given two or three times dai until the tetany is relieved.

'Conditioned' Calcium Deficiencies

Apart from a defective intake of calcium in the diet, osteoporo may be met with in practice in conditioned deficiency stat where calcium metabolism is defective even in the presence an otherwise adequate intake. The most common causes of su conditioned calcium deficiencies are:

(1) *Factors interfering with the Absorption of Calcium.* The mc striking manifestation of these is the osteoporosis th frequently occurs as a complication of steatorrhœa. In tl condition the calcium deficiency may be so severe as result in a fall in the serum calcium level and the appearan of tetany. Chronic inflammatory lesions like ulcerati colitis also interfere with calcium absorption.

(2) *Endocrine Disturbances.* While hyperparathyroidism is tl most striking example of an endocrine disturbance causi skeletal decalcification, overactivity of other endocri glands may produce lesser degrees of this condition, ar osteoporosis may be found in thyrotoxicosis, acromega and the syndrome produced by basophil adenoma of tl pituitary.

(3) *Renal Disease.* Where renal insufficiency has reached tl stage at which excretion of phosphorus by the kidney interfered with, the amount of phosphorus excreted by wa of the bowel is increased. This extra phosphorus in tl intestine unites with calcium to form insoluble calciu phosphate and so renders it unavailable. Renal rickets the name applied to the extreme bony lesions that resu from long-standing renal insufficiency in children, but it now recognized that the symptoms and signs of osteoporos may develop in adults suffering from renal disease.

II. Phosphorus

This element is widely distributed in the animal body, where performs many metabolic functions. All compounds of pho phorus in biological material appear to be derivatives either orthophosphoric acid (H_3PO_4) or pyrophosphoric acid (H_4P_2O Derivatives of orthophosphoric acid are the inorganic phosphat in the bones, teeth and body fluids; and organic forms combined with fat (*e.g.* phosphatides such as lecithin), with protein (*e.g.* casein and nucleoprotein), and with carbohydrate (*e.g.* hexosephosphates, important in the chemistry of muscular contraction). Pyrophosphoric acid occurs in certain of the co-enzymes that play an important part in the intermediary metabolism of carbohydrate – namely, adenylic acid pyrophosphate and co-carboxylase (aneurin hydrochloride pyrophosphate). The body is apparently able to synthesize all the organic compounds of phosphorus it requires from inorganic phosphates and the appropriate organic material.

In general the utilization of phosphorus is influenced by the same factors as those which affect the utilization of calcium. The importance of the ratio of calcium and phosphorus in the diet in the absorption of these elements has already been discussed on p. 128. Apart from calcium, there are many other cations which form insoluble compounds with phosphates under the conditions existing in the alimentary tract, and so interfere with the absorption of phosphorus, *e.g.* iron, aluminium, lead, beryllium and manganese. This is, however, mainly of experimental interest.

Dietary Requirement

The requirements of the body for phosphorus have not been investigated so fully as those for calcium, but they have been assessed by essentially the same criteria. About 99 per cent of the calcium in the body is present in the skeleton, the greater proportion of which is combined with phosphate, but phosphorus is required for numerous purposes in the body in addition to the formation of bones and teeth, and some 20–30 per cent of the body's total phosphorus content is present in the soft tissues unassociated with calcium. Phosphorus is therefore at least as vitally important a mineral as calcium. Owing to its wide distribution in foodstuffs, especially those of animal origin, there is, however, less risk of deficiency of phosphorus in the diet than of calcium, and unless calcium is being taken medicinally in large amounts as the chloride, lactate or gluconate, there is little chance of the phosphorus intake ever lagging behind.

In experimental animals, diets very low in phosphorus and high in calcium produce rickets, even in the presence of abundant vitamin D. The effects of a diet deficient in phosphorus, but adequate although not excessive in other respects, has been little studied, but in a recent report it is stated that young rats fed on such a diet show a defective rate of growth followed by deat In an effort to make good the dietary deficiency, phosphorus mobilized by the breaking down of the calcium phosphate of th bones, and is transferred to the soft tissues for the purpose c promoting their growth, the calcium being excreted. This lead to progressive severe demineralization of the bones, with spor taneous fractures and collapse of the bony thoracic cage. Simila conditions have been reported among farm animals reared o herbage from phosphorus-poor soils. The level of inorganic phos phorus in the blood serum falls, and severe demineralization c the bones occurs, with a relative increase in the proportion c calcium carbonate to calcium phosphate in the bones.

The minimum daily amount of phosphorus necessary to preserv phosphorus equilibrium in a normal adult is stated to be 0·9 gram If the same 50 per cent margin of safety is allowed as was don in the case of calcium, this gives a requirement of roughly 1·3 gram of phosphorus daily. In pregnancy and lactation, this intak should be doubled, and for children it should be increased b half or doubled, according to the age of the child.

Phosphorus Content of Foodstuffs

As stated above, flesh foods are rich in phosphorus, all of whic is in a form that can be utilized by the body. Some of the plan foods also contain a large amount of phosphorus, for exampl the legumes and cereals, but a large and variable proportion o the phosphorus in these foods is present in the form of phyti (see p. 129), and therefore cannot be utilized efficiently by th body. Some of this phytin is, however, broken down by bacteria action in the intestines and its phosphorus thus rendered availabl It is safe to assert that, in general, if the calcium requirement of a diet are adequately met, that diet will supply sufficien phosphorus.

III. Iron

Although present in very small amounts in the body (abou 6 grams), iron is one of the most important elements in nutrition and is of fundamental importance to life. It is essential to th formation of hæmoglobin, the hæmatin radicle of which contain about half the total iron of the body. Traces of iron exist i practically every tissue of the body, where iron-containing com ds like cytochrome play an important part as catalysts i damental processes of intracellular oxidation. The amoun of iron present in this form is small, amounting to not more than 0·3 gram for the entire body. The amount of non-hæmoglobin iron in the different tissues is relatively constant, and does not decrease in experimental animals even after prolonged and severe anæmia. It is therefore doubtful if the processes of intracellular oxidation that depend on the presence of this iron are ever interfered with by a lack of iron in the diet, and it has been suggested that the needs for this tissue iron are met by the body before iron is utilized in the formation of hæmoglobin. The rest of the iron in the body is present as a reserve store in the liver, and to a lesser extent in the kidney, spleen and other organs, and as a working supply in the manufacture of hæmoglobin of which some 25 grams, containing about 90 milligrams of iron, are formed daily by a normal adult to replace that destroyed in the breakdown of effete red blood cells.

Absorption and Availability of Iron

It has been stated that absorption of iron can take place from the stomach and throughout the whole of the small intestine, but the greatest absorption occurs in the upper part of the small intestine. Iron is not very readily absorbed, and an apparent prerequisite is that it should be in soluble, ionizable form. Inorganic salts of iron are effective under experimental conditions as the sole source of dietary iron in the relief of iron-deficiency anæmia, while organic forms of iron in complex compounds such as hæmatin, which are not broken down in digestion, are not available to the body. It follows therefore that inorganic iron is superior as a source of iron, and that the iron content of foodstuffs as determined by chemical analysis does not necessarily represent the biological availability of this iron to the body.

A method of estimating the 'available' iron in foods was devised by Hill (1930); this depends on the property of an organic reagent, αα-dipyridyl, which reacts with ionizable ferrous but not ferric iron. It has been claimed that in experimental animals there is a fair agreement between the availability of iron in foodstuffs as measured by Hill's method and by their effectiveness in relieving iron-deficiency anæmia, and that Hill's test is therefore a measure of the biologically available iron in foodstuffs. Several workers, however, deny this, and contend that the biological availability of iron in foodstuffs may differ from the value obtained by this estimation. In the present state of uncertainty regarding the assessment of the biological availability of iron for man, it is probably wiser to consider the iron content of th diet and the dietary iron requirements in terms of total iron, wit the reservation that a certain proportion of the iron of the die may not be in a form that the body is capable of absorbing. I is of interest to note, however, that on the basis of estimatio with αα-dipyridyl, about 60 to 70 per cent of the iron of a average mixed diet is present in inorganic ferrous form an therefore available to the body.

Two other important factors in the absorption of iron must b mentioned, namely the effect of the calcium : phosphorus rati and of achlorhydria. In experimental animals a diet low i calcium and high in phosphorus leads to the development of a iron-deficiency anæmia. This can be prevented by the additio of calcium or other cations such as beryllium which combin with phosphorus in the intestine to form insoluble phosphate showing that the cause of the anæmia produced by diets with low calcium : phosphorus ratio is the interaction of the exces phosphorus with iron to form an insoluble phosphate and s render the iron unavailable. This influence of calcium and phosphorus on iron absorption is of more than academic interest, fo one of the authors (Davidson *et al.*, 1933) has shown clinicall that a normal blood picture can be maintained on a diet containing only a moderate amount of iron provided it is also ric in calcium, whereas the same iron intake leads to the developmen of anæmia if the calcium content is reduced considerably.

The presence of hydrochloric acid in the gastric juice is evidently important for the solution of food iron and the reductio of ferric iron to the ferrous state, since achlorhydria affects iro absorption adversely and may lead to anæmia, possibly throug failure in the liberation of iron from protein.

Unlike most of the other nutrients, iron is not excreted eithe by the kidneys or the intestinal tract in appreciable quantities the body stores of iron being utilized again and again on thei liberation from organic combination. Iron metabolism as far a absorption and excretion are concerned is therefore a 'one way process in that iron is either absorbed and retained or if no absorbed, eliminated in the fæces. According to McCance an Widdowson (1937), absorption of iron from the intestine depend on the ratio between the iron content of the intestinal content and that of the plasma, and where the latter is normal, practically no absorption of iron will occur. This conception of iron metabolism explains why some workers have been able to maintain healthy adult males in iron equilibrium on diets containing as little as 1 milligram of iron per day. It is therefore extremely unlikely that iron deficiency in human beings can ever result solely from ingestion of a diet very low in iron. Clinical observations support this view, for careful investigation of cases of iron-deficiency anæmia responding to iron administration will practically always reveal some source of blood loss or other pathological condition to explain the iron deficiency. After absorption, iron is carried in the plasma and is apparently taken up by the liver, where the greater part is stored. Smaller stores of iron are also found in the spleen, kidney, and other organs.

Dietary Requirements

The requirements for iron vary with age and sex to an even greater degree than for most of the other nutrients. There are two sources of iron in the body: first, the child is born with an initial supply which it has received from the mother during intra-uterine life; secondly, after birth, iron is obtained from the diet. At birth iron is stored in the tissues and also in the excess circulating hæmoglobin of the physiological polycythæmia which is then present. Both human milk and cow's milk contain very little iron, so that for some months after birth the child is dependent on the stores present in the organs and on that derived from the hæmoglobin liberated during the physiological hæmolysis which occurs in the first few weeks after birth. The fœtus usually obtains an adequate supply even at the expense of the mother, but this process is not unlimited, and unless the mother's intake and store are adequate, the iron reserves of the infant are likely to be deficient. The increasing demands for the rapid growth that occurs after birth, coming as it does when the infant's diet is low in iron, soon exhaust such reserves, and anæmia develops. These factors help to explain the findings of one of the authors (Davidson *et al.*, 1935), that in infants under two years belonging to the poorest classes in North-East Scotland iron-deficiency anæmia was present in no less than 41 per cent of the large number examined. It has, however, been suggested recently that, of the stores available after birth, the iron derived from physiological hæmolysis is more important than that which is stored in the infant's organs and tissues. Infants with a low birth-weight have a smaller blood volume than normal infants, and birth-weight may therefore be of more importance in determining the development of anæmia in infants than the state of the maternal iron reserves.

Whatever the actual mechanism in the production of ir deficiency anæmia in infants during the first year or two of li such anæmia is liable to persist into childhood unless treate and in the study by one of the authors quoted above, anæm of the iron-deficiency type was found in 32 per cent of pre-scho children. The increasing intake of iron that accompanies t greater and more varied diet eaten by the growing child evidently just sufficient to meet the increasing demands for growt but cannot make good the iron deficiency with which the child w born, so that the subnormal hæmoglobin level arising in infan persists unless raised by the therapeutic administration of iron.

When describing iron metabolism it was stated that the excr tion of iron is for all practical purposes negligible so that adult provided they reach full growth with a normal hæmoglobin leve should not develop iron-deficiency anæmia, however small the intake of iron, in the absence of any loss of iron from the boc due to hæmorrhage or to its transfer to the fœtus. This explai why iron-deficiency anæmia is to all intents and purposes unknow in adult males except as a conditioned deficiency resulting fro abnormal blood loss which may occur from chronic and subt causes such as bleeding hæmorrhoids, oozing from a chronic pept ulcer, intestinal infestations, *e.g.* hookworm disease, that deman careful examination to reveal the source of the hæmorrhag Failure in absorption of iron as a result of pathological states the gastro-intestinal tract is a less common cause of anæmia.

In women, however, the recurrent loss of iron in the menstru blood flow must be made good from the diet if anæmia is to b avoided, quite apart from the added drain on the body's iro stores imposed by pregnancy and lactation. The iron loss a menstruation varies considerably among different individual According to the investigations of Barer and Fowler (1936), th amount at each menstrual period varies in normal women fro about 3 to 90 milligrams, the average loss in their series of cas being 25 milligrams. On the other hand, the same workers foun that the average loss in a group of anæmic women was muc higher, namely 120 milligrams. Other workers have recorde figures similar to those of Barer and Fowler for normal wome for instance, in four young women whose iron metabolism wa studied over several menstrual circles, Leverton and Robert (1936 and 1937) found that the average menstrual losses of iro varied from 14 to 23 milligrams, the losses being approximatel constant over successive periods for the same subject.

There seems little doubt that even normal menstruation imposes a strain on the iron resources of the body unless a diet with a generous iron content is taken. In the four young women studied by Leverton and Roberts, the hæmoglobin levels at the beginning of the metabolic experiment ranged from 84 to 94 per cent, whereas after several months on an average daily iron intake ranging from about 10 to 14 milligrams, the hæmoglobin levels rose to 94 to 103 per cent.

Similarly, Widdowson and McCance (1936) found that the average daily intake of iron by English middle-class women on freely chosen diets which were not limited by economic circumstances was in the neighbourhood of 11 milligrams. Although these women had normal hæmoglobin levels according to the current standard for women, administration of iron in therapeutic doses raised their hæmoglobin values by 4 to 17 per cent.

The evidence presented above suggests that the general finding that the average hæmoglobin level in normal women is lower than in men is not an expression of any physiological difference between the sexes, but rather an indication that the diet of the average woman contains insufficient iron to maintain optimum levels of hæmoglobin. An arbitrary calculation based on Widdowson and McCance's findings will show how near even an adequate diet is to the danger limit. It has been estimated that roughly one-half of the available iron in the diet may be absorbed when there is a demand for iron on the part of the body, and if one assumes that about 65 per cent of the total iron of an average mixed diet is available to the body, then the amount of iron that could be absorbed daily from the pre-war diet of the average Englishwoman would be 11 × 65 per cent ÷ 2 = 3·6 milligrams. Provided this maximal rate of iron absorption was maintained continuously, the iron retention over a month would be about 108 milligrams, which is enough to cover the maximal normal menstrual losses recorded by Barer and Fowler, but inadequate to prevent progressive loss of iron and the development of anæmia in the group of women with menorrhagia and an average menstrual loss of iron of as much as 120 milligrams.

Unfortunately, many of the iron-rich foods, for example meat, are expensive, and the pre-war diet of women of the poorer classes in a city in Scotland was shown by Fullerton (1936) to contain on the average only about 7·5 milligrams of iron per day. If one applies the same calculation to this figure as was made in the case of the iron intake of middle-class Englishwomen, it will be found that the monthly iron retention would only amou to about 72 milligrams, which is inadequate to meet the high range of normal menstrual iron losses reported by Barer ar Fowler, let alone the losses they found in anæmic women. It not surprising therefore that in the large-scale investigation b one of the authors already mentioned (see p. 141) approximate 50 per cent of the adult women of the poorest classes in tl north-east of Scotland were suffering from some degree of anæm of the iron-deficiency type.

While it is true that some of the iron-rich foods are expensiv it does not follow that persons whose diet is limited by econom circumstances must perforce suffer from a low intake of iro As described in Chapter XIX, and shown in tabular form Table 11 on p. 380, it was possible before the war to constru a generally satisfactory diet containing about 13 milligrams iron per day at a cost of only sevenpence.

The demand for iron caused by pregnancy and lactation ha been represented by Davidson and Fullerton (1938) thus:

Iron	content	of fœtus at term . . .	400 mg.
,,	,,	of placenta and uterus . .	150 ,,
,,	,,	of blood lost during delivery .	175 ,,
,,	,,	of milk during 6 months' lactation	180 ,,
			905 ,,

Over the 450 days of pregnancy and lactation, this represents a average daily loss of iron from the maternal resources of 2 mill grams, which is over twice the average loss reported by Bar and Fowler in normal menstruation. It is clear, therefore, tha the diet must supply generous amounts of iron if iron-deficienc anæmia in the mother is to be avoided. There is normally progressive fall in hæmoglobin level during pregnancy, but th anæmia usually improves abruptly within a few days of deliver and this so-called 'physiological anæmia' is actually a hydræmia In view of the conditions regarding hæmoglobin levels in non pregnant women mentioned above, it is not surprising, howeve that several workers have shown that the hæmoglobin levels o many pregnant women may be raised significantly by the thera peutic administration of iron, indicating that iron deficiency ma often be a causal factor in the anæmia of pregnancy in additio to hydræmia. A more striking indication of the strain impose by pregnancy on the maternal iron resources is the finding tha in adult women the incidence of anæmia is higher among thos who have borne children than among nulliparæ, and that the severity of the anæmia depends in some measure on the number of children borne.

The important effect of diet on the incidence of iron deficiency in this country has been demonstrated in a recent report by one of the authors (Davidson *et al.*, 1944), in which it has been shown that in Edinburgh there was a progressive rise in the hæmoglobin levels of schoolchildren and pregnant women during 1943 and 1944, in spite of the exigencies of war-time rationing. This improvement is ascribed to the introduction in 1942 of national flour with its higher iron content than that of white flour, a measure which was largely responsible for the increase in the amount of iron in the foodstuffs available for consumption *per capita* in Great Britain from 13 milligrams per day in 1938 to 16 milligrams per day in 1943 (*Food Consumption Levels*, H.M. Stationery Office, 1944).

The most recent estimates of the desirable total iron intake at all ages and in both sexes are given in Table 1. These figures are of course much greater than the actual physiological needs of the body for iron, since they make allowance for the fact that only a relatively small part of the iron of the food is absorbed. Theoretically, the iron requirements of the normal male are doubtless lower than the figure stated, but there is much to be said for the intake of a generous amount of iron in males, as well as females, as a means of protection against any drain imposed on the iron resources of the body by hæmorrhage. As has been shown, an adequate intake is of much greater importance in females, and the figures given in Table 1 do not err on the side of generosity, especially in the case of pregnancy. Some workers have found that an intake of 15 milligrams of iron per day is barely sufficient to maintain a positive iron balance in pregnant women and recommend that the desirable daily iron intake should be as high as 20 milligrams per day throughout the child-bearing period.

The question of the foods that will best make up the desirable iron intake is complicated by the factor of the availability of iron in foodstuffs, already discussed. In view of the uncertainty regarding the availability of iron in foodstuffs to man as distinct from experimental animals, consideration is being given here mainly to the total iron content of foods. Among the richest sources of iron are liver and the other edible organs and black treacle. Next in order of iron content come nuts, muscle meats, wholemeal cereals and dried pulses. Although both cereals pulses have a lower iron content than muscle meat when coo it is probable that they are at least as good sources of iron to body in view of the fact that some of the iron in meat is contai in myohæmoglobin, and is therefore not available to the b By the dipyridyl method the availability of the iron in mu meat is only 25 per cent or less. Eggs, nuts, prunes, raisins, d apricots and peaches, chocolate and certain of the leafy vegetal especially spinach and turnip tops, are also relatively good sou of iron; the availability of the iron in spinach is, however, than in other leafy vegetables, as judged by estimation w dipyridyl. Milk is conspicuously low in iron, hence the importa in the first few months of the store of iron with which the in is born.

An unpredictable factor in relation to the dietary intak iron is the amount that is added to the food in preparation cooking as a result of contamination from iron utensils, kni etc. This explains why the actual iron content of a dietary san subjected to analysis is often found to be greater than theoretical value calculated from tables of food values, in wh the figures for iron are of course based on analyses carried with special precautions against such contamination.

It is important to realize that diet alone should never be re upon in the treatment of established iron-deficiency anæn since it can barely replenish a store that has been depleted to point of producing anæmia. It has indeed been estimated t the absorption and utilization of 5 milligrams of iron daily, wh demands a diet with a generous iron content, will raise hæmoglobin level only about 6 per cent in a month. Curat treatment of iron-deficiency anæmia therefore demands the the peutic administration of iron salts. This is discussed in grea detail in Chapter XIX.

IV. Iodine

Iodine, although present in such small amounts in foodstuffs to be classified among the 'trace' substances, is essential normal growth, development and health, being of particu importance in relation to thyroid function. Man, like ot animals, must obtain iodine in the required amounts from food, and if the supply is inadequate, pathological effects iodine deficiency – goitre and cretinism – result.

In the body the only known function of iodine is to take p with the amino-acid tyrosin in the formation of thyroxin, the active principle of the thyroid gland. Thyroxin is present in a combined form with globulin as the complex substance thyroglobulin, which appears to be the true active secretion of the thyroid gland and also contains diiodotyrosin, an intermediate substance in the formation of thyroxin.

The total amount of iodine in the body of an adult man is very small, probably about 50 milligrams. Iodine occurs in all the tissues of the body, but only in traces except in the thyroid gland, which, with an average concentration of iodine of about 0·2 per cent (dry weight), contains roughly one-fifth of the total iodine of the body. The thyroid absorbs iodine from the blood and converts this rapidly into thyroglobulin, which is stored in the colloid material in the alveoli of the gland. Once these stores in the thyroid gland are full, any excess iodine is excreted in the urine, fæces, tears, etc. The maximum amount of iodine required to maintain the thyroxin stores in the thyroid gland is said to be about 0·2 milligram per day, but some of the iodine freed from broken-down thyroxin is used by the thyroid gland in the manufacture of fresh supplies, in a manner similar to the repeated use of iron in the formation of hæmoglobin, so that the daily iodine requirements are less, and have been estimated at about 0·05 milligram for adults, and greater amounts, in the region of 0·15 milligram, for children.

Iodine is widely distributed throughout nature, occurring in many forms, both organic and inorganic, in almost all living things, which derive it ultimately from the soil and water. The iodine content of foodstuffs and of water depends on the iodine content of the soil on which foods are produced and through which the water passes. There are wide variations in the iodine content of soils in different areas of the earth's surface. In certain areas the soil is very deficient in iodine; here the foodstuffs and water will have a very low iodine content, and the iodine intake of the inhabitants of such areas will therefore be inadequate, with the result that the pathological effects of iodine deficiency will be endemic there. In other regions, near the sea for example, the soil and water may be rich in iodine. Sea water contains a relatively high proportion of iodine, so that marine products, both vegetable and animal, are rich sources of iodine. Areas where the iodine content of the soil and water is especially low, and iodine deficiency is therefore endemic among the inhabitants, are chiefly, but not exclusively, mountainous regions – *e.g.* the Alpine regions of Southern Europe (including the Balkans), the H layan regions and the high plateaux of Western China and Eas Mongolia, the Andes, the Rocky Mountains and the basin of Great Lakes in America. In Great Britain the most impor goitrous areas are in Derbyshire and the Thames Valley.

Effects of Iodine Deficiency

The two pathological conditions resulting from iodine deficie goitre and hypothyroidism, may occur together or separat Hypothyroidism due to iodine deficiency is, however, usu accompanied by a goitre; in its most severe form it leads to condition known as cretinism. Goitre due to iodine deficienc usually endemic, but sporadic cases may occur in other t goitrous areas due to factors which lead to a conditioned deficie of iodine. On the other hand, hypothyroidism due to iodine ficiency is for all practical purposes entirely endemic in its i dence. Sporadic cases of cretinism are almost always athyro for some unknown congenital reason, while adult hypothyroid (myxœdema) is practically always due to causes other than io deficiency.

Simple Goitre. Simple goitre is the name given to the enla ment of the thyroid gland that occurs when the available sup of iodine is reduced and the gland undergoes hypertrophy hyperplasia in an effort to obtain sufficient iodine to keep up stores of thyroxin. Such enlargement of the thyroid does necessarily indicate that the secretion of its hormone is defici On the contrary, although the gland is overworked and has hypertrophy in an effort to maintain the normal supply of thyro this is achieved. Hence simple goitre is not accompanied signs of hypothyroidism until the supply of iodine beco absolutely as well as relatively deficient, and the supply hormone to the tissues becomes insufficient. The stage of failur the supply of thyroxin rarely occurs except when iodine deficie persists over several generations; in such circumstances child are born with already subnormal thyroid glands, and the tende to goitre increases progressively with each succeeding generat until cretinism results.

Like other deficiency diseases, simple goitre may be due to absolute lack of the essential nutrient, in this case iodine, or m be the result of a conditioned deficiency where increased requi ments or perhaps failure of absorption of iodine make an apparen normal intake of this substance inadequate. Factors increasi the demand for thyroxin, and hence for iodine, are bodily growth, puberty, pregnancy and probably menstruation. With a liberal supply of iodine in the diet, the extra demands can be met without difficulty, but in the presence of a borderline supply of iodine any of them may precipitate an iodine deficiency. This explains the greater incidence of simple goitre in children and in females. Another secondary factor in the production of iodine deficiency has been demonstrated in laboratory animals, although its effect n man, if any, is unknown. It has been found that simple goitre can be produced in animals by feeding them on a diet consisting solely of cabbage. Although the goitrogenic substance in cabbage has not yet been identified with certainty, it is thought that the organic cyanides in cabbage depress oxidation in the tissues and o create an increased demand for thyroid hormone to counteract his effect.

Pathology of Goitre. The pathological appearance of goitre due o iodine deficiency is at first a hypertrophy and hyperplasia, with enlargement and increased vascularity of the gland. The pithelium changes from low cuboidal to columnar, the alveolar walls become infolded and project as processes into the lumen, nd the colloid decreases in amount. The thyroid hyperplasia of puberty or pregnancy presents a similar appearance. The next stage in the pathological picture is either one of atrophy, if the deficiency of iodine persists, or involution, if the supply of iodine ecomes adequate again either on account of an increased intake r the cessation of the condition causing a relative deficiency. n atrophy of the thyroid the alveolar cells become irregular and egenerate, the colloid decreases and the stroma of the gland ecomes relatively or absolutely increased. More commonly, owever, involutional changes, which are a reversal of those of the hyperplastic stage, follow. The blood supply of the gland ecreases, the epithelium shrinks to low cuboidal and often attened cells, and colloid again fills and even over-fills and istends the follicles. If involution is arrested at this stage, the nlarged gland with distended, colloid-filled follicles forms what is known as a colloid goitre, but in a certain proportion of cases involution is more complete and the gland returns to normal as ar as macroscopic appearances go.

If this process of hyperplasia and involution is repeated, as it may be by a recurrence of conditions leading to a relative iodine eficiency, involution is liable to be less complete and each time the goitre is left a little larger. Incomplete involution is apt to become localized, and nodules either of hyperplastic or col distended follicles may develop, often becoming surrounded dense fibrous tissue, or going on to cyst formation or degenera calcification. Nodular goitres are prone to cause symptoms to local pressure on the trachea and the veins or nerves of neck, and there is also a marked tendency to malignancy. It m be emphasized that all these changes in the thyroid gland without general effect on the body, except in persons wh ancestors for several generations have been subject to io deficiency, in whom the thyroid undergoes atrophy instead involution. This is the sequence of events in cases of ende cretinism, where the thyroid may vary in size from one m smaller than normal and composed almost entirely of fibr tissue to a large goitre with the changes found in a simple goi The other changes in cretinism are due to retarded developm and include stunted growth, delayed calcification of the skele and union of the epiphyses, delayed dentition, and incomp development of the brain.

Treatment of Iodine Deficiencies

More than in any other deficiency disease, prophylactic treatm of iodine deficiency is conspicuously more successful than cu tive treatment.

Preventive treatment by the administration of iodine in form or another has been applied in many of the goitrous ar of the world with striking effects on the incidence of simple goi and cretinism in these regions. Iodine prophylaxis is usua accomplished by adding traces of potassium or sodium iodide table salt (in America sufficient is added to give an iodine c centration of 1 : 100,000); alternatively, the administration three grains of potassium iodide daily for ten days, or one dr of Lugol's solution daily for three or four weeks in spring a autumn is equally effective.

Such general preventive measures are sufficient also to m the increased demands for iodine that occur at periods of phys logical stress, such as puberty and pregnancy. In areas wh the dietary iodine supply is liberal, protection of special grou of the population may not be necessary, but in borderline regi where there is any reason to suspect that the iodine content the diet may be low, protective treatment is advisable duri puberty (a drop of Lugol's solution twice or thrice weekly two or three weeks in spring and autumn) and pregnancy (

drop of Lugol's solution per week during the last half of pregnancy).

Objections have been raised to the general use of iodized salt against iodine deficiency on the grounds that such treatment would be apt to induce thyrotoxicosis in persons with already overactive thyroid glands, or in those with nodular simple goitre. There is no very convincing proof that hyperthyroidism commonly results from the administration of iodine to subjects with simple goitre, although when the use of iodized salt became general in the State of Michigan, U.S.A., an increase in the number of cases of nodular toxic goitre coming to operation was noted for the first two years, this temporary increase being succeeded, however, by a rapid and steady decrease.

When iodine deficiency has led to the development of a goitre, treatment with iodine is much less successful than in the prevention of goitre. Small goitres may sometimes lessen in size, or even practically disappear in young people, on treatment with iodine in larger doses than those used for prophylaxis (*e.g.* 1 to 5 drops of Lugol's solution daily for a month, this being repeated in alternate months over a year). Goitres of large size and long duration have usually reached the colloid stage, and these respond poorly if at all to iodine, the same being true of nodular goitres, in which the nodules may even be accentuated as a result of treatment. Thyroid extract is no more effective than iodine in the treatment of simple goitre, and should not be prescribed unless there are associated symptoms of hypothyroidism – a rare occurrence except in areas of endemic goitre.

Pressure symptoms caused by simple goitre may demand surgical intervention, but surgery should not be employed merely for cosmetic reasons in young patients until adult life is reached.

Sodium and Chlorine

These elements require consideration here, not only because a dietary deficiency may occur in normal individuals due to excessive losses in the sweat, but also because the amount of sodium chloride in the diet must be controlled in certain diseases.

Some sodium takes part in the structure of cartilage and muscle cells, but the greater part of the sodium of the body is present in the blood and interstitial fluid, largely combined with chloride and bicarbonate, where it plays an important part in the maintenance of the osmotic pressure and the acid-base equilibrium of these fluids. Chlorine is present in all the body tissues and fluids, almost entirely in inorganic form as chlorides. The chloride the blood and interstitial fluid, particularly sodium chlo form about two-thirds of the anions of these fluids, where t have an essential rôle in neutrality regulation and serve in g measure to maintain their osmotic pressure. Chlorides are required to provide the hydrochloric acid of the gastric juic

The internal secretion of the cortex of the adrenal gl controls the sodium content of the body fluids, possibly by influence on the reabsorption of sodium (and secondarily chlor from the glomerular filtrate in the renal tubules. In adr insufficiency there is evidently a failure in this reabsorption, the excessive excretion of sodium in the urine leads to a nega sodium balance, a fall in the level of sodium chloride in the bl and a consequent reduction in blood volume.

Most foodstuffs, especially those of plant origin, contain m more potassium than sodium. In unconscious compensation this imbalance between the two elements in his diet, and enhance its palatability, man from time immemorial has b accustomed to add salt to many of the foods he eats. Un ordinary circumstances both sodium and chlorine are supp abundantly in the food and the salt added to it in cooking at table. The minimal daily requirement of sodium chloride maintain equilibrium in an adult is below 2 grams, which is m less than the average intake of 10 to 15 grams when salt is ad in the usual amounts to the food.

Sodium Chloride Requirements at High Temperatures

The predominant effect of a high environmental temperature the physiological processes of the body is the added difficu imposed on the mechanism for getting rid of the heat produ in metabolism. When the air temperature reaches or exce that of the body, heat loss by radiation and conduction is longer possible, and the only important channel of heat loss l to the body is by the evaporation of sweat. Heat production the body is not lessened in a hot climate except by a volunt reduction in physical activity, and possibly by the slight red tion in basal metabolism (up to 10 per cent) that has sometin been recorded among natives of the tropics or Europeans w have lived there for some time. The burden of heat dissipation high environmental temperatures therefore falls largely on t sweat-secretory mechanism.

Sweat consists essentially of a dilute solution of sodium chlori sodium lactate and a small amount of urea. The excretion of water in the sweat is of course essential for the dissipation of heat (the evaporation of one litre of water at a skin temperature of 91° F. makes latent about 580 Calories), and the elimination of urea assists in a small way the function of the kidneys in getting rid of this substance. But the sodium salts and the lactic acid in the sweat serve no known physiological purpose and their loss is expensive to the body. The concentration of sodium chloride in the sweat varies considerably among different individuals under comparable conditions, and this difference cannot be related to any known physiological circumstance such as age, physical condition or properties of the blood.

Not only does the concentration of salt in the sweat vary among different individuals, but in the same person the concentration may differ under varying conditions. The salt content of the sweat is stated by some workers to vary with the body temperature and with the rate of sweating. Thus the greater the rate of sweating or the harder the work, the greater the salt loss per unit volume of sweat. The composition of the sweat bears little relation to minor variations in the water and salt intake, but the salt content of the sweat is decreased when the salt intake is reduced to levels little above the minimum requirement of 2 grams per day, or where salt loss has brought about a significant fall in the serum chloride level. The sweat glands therefore do appear to be able to respond protectively to inadequate salt intake or excessive salt loss, but this mechanism is imperfect, and salt loss continues in the sweat even under these unfavourable conditions, thereby producing a negative salt balance.

The bodily changes that lead to acclimatization to high temperatures are not yet fully understood, and the major factor is probably circulatory rather than metabolic. But one manifestation of acclimatization is a decrease in the concentration of salt in the sweat occurring within a few days of exposure to work at high temperatures.

In a summary of the reports of different workers compiled by Dill (1938) the range of sodium chloride concentration in the sweat varies from 0·03 gram per 100 c.c. to 0·8 gram per 100 c.c., but Dill states that in his experience the concentration in acclimatized individuals over a 24-hour period rarely exceeds about 0·15 gram per 100 c.c. Even at this low level, however, the loss of salt in the sweat of persons living and working in a hot climate may be considerable owing to the large volumes of sweat secreted;

the total sweat production over 24 hours in persons with acce to an unlimited water supply may be anything from 6 to 8 litr under conditions of moderate activity, and up to 10 to 15 litr where hard physical work is being undertaken. The latter swea volumes would lead to a loss of 15 to 22 grams of salt per da and in persons who habitually secrete sweat with a high salt co centration the loss will be much greater – in the region of 30 gram per day or more.

It is evident therefore that the usual salt intake of 10 to 15 gram per day may be inadequate to meet the requirements of perso living and working in hot climates, and unless special precautio are taken to increase the salt intake, sodium chloride deficienc will result. The earliest manifestations of this deficiency diseas are weakness, excessive fatigue, anorexia, and nausea leading t an impairment of physical performance and a diminution i mental acuity. If salt loss is accompanied by fluid loss, as i usual when strenuous activity is being undertaken, these symp toms are aggravated by the development of painful spasms the voluntary muscles, the so-called 'heat cramps', and examina tion of the blood will show a reduction in the level of sodiu and chloride ions in the plasma and the usual evidence of deh dration – an increase in the red cell count and the level of th plasma proteins.

As we have seen, the sweat glands have only a limited powe of conserving salt, and will go on excreting it even when th body store is depleted. The kidneys, however, attempt to co serve sodium chloride by reducing its concentration in the urin and individuals whose body stores of salt are depleted excret urine containing little or no chloride. There is unfortunately n craving for salt even when it is badly needed – man appears t have lost this natural instinct that is seen in most species animals.

The osmotic pressure of the blood is not reduced in sodiu chloride deficiency, thirst and renal activity being adjusted s that the extracellular water of the tissues is reduced. This lead to the most dangerous of the sequelæ of sodium chloride deficienc namely dehydration and a lowering of the blood volume whic may be referred to as secondary dehydration to distinguish i from primary dehydration due to simple water lack. Unlike th latter, secondary dehydration is not accompanied by thirst an may thus progress insidiously until the reduction in blood volum precipitates heat exhaustion and circulatory failure.

Treatment of Sodium Chloride Deficiency

The prevention and treatment of sodium chloride deficiency is theoretically a relatively simple matter. It must be remembered, however, that this condition is insidious in its onset and is accompanied neither by thirst nor by a craving for salt. In the absence of the only specific symptom, the muscular cramps, diagnosis must be made on the occurrence of the indefinite symptoms mentioned above under conditions where salt deficiency might be expected, the virtual disappearance of chlorides from the urine, and the blood findings if the case is an advanced one.

Prevention of salt deficiency is achieved by increasing the salt intake of persons exposed to high temperatures, either in hot climates or in industrial conditions, such as the stokeholds of ships or in mines. The extra salt can be added to the food, but reliance should not be placed on this measure alone, for some persons may not care for highly salted food or may eat less for various reasons. A more certain and more physiological method of increasing the salt intake is to add salt to the drinking water. The concentration recommended is 0·1 gram per 100 c.c., which is slightly less than the average salt content of the sweat, and is only slightly saline to the taste if the salted water is cool when drunk. The taste of salt in water is also well disguised in hot beverages like tea. The salt ingested in the drinking water, together with that consumed in the food, will make good the salt loss in the sweat. Salting of the drinking water has the great advantage that the intake of salt is thereby automatically adjusted to the amount of water drunk which in turn is roughly approximate to the amount of sweat lost. Where there is no convenient central point at which salt can be added to the water, as may occur under conditions of warfare in hot climates, tablets containing 10 grains of sodium chloride should be made available, manufactured in such a way that they will dissolve freely in water. One of these tablets added to each pint of water drunk will give a salt solution of approximately 0·1 per cent strength.

The recommendation that salt should be added to the drinking water must be qualified by stating that it is applicable only to circumstances where there is access to an unlimited water supply. Where the water intake is restricted, the amount of sweat secreted, and hence the loss of salt, is less. Under these circumstances the amount of salt usually taken with the food will be adequate to meet the salt loss in the sweat, and extra salt taken with the drinking water will have to be excreted by the kidneys, carryin with it water which the body needs urgently.

The treatment of sodium chloride deficiency is simple an consists in the administration of sufficient salt to make good th depleted body stores. This can be achieved most rapidly by th parenteral injection of normal saline, or more slowly by givin the patient one-third normal (0·3 per cent) saline to drink. A this strength saline is not unpalatable, especially if flavoure with fruit juice, and will not cause emesis. It cannot be to strongly emphasized that prostration due to salt deficiency shoul not be treated by the administration of plain water. Water alon cannot be retained for long by a salt-depleted patient, and henc will not relieve secondary dehydration; instead it is excrete through the kidneys and the skin in order to maintain the constancy of the osmotic pressure of the body fluids. If it is retaine it lowers the osmotic pressure of the body fluids, which cause violent muscle cramps, nausea and vomiting and makes th patient seriously ill.

Sodium Chloride in Medical Dietetics

The importance of sodium chloride in the dietary treatment o disease may be summarized as follows:

(1) It must be restricted in the diets given to patients wit congestive heart failure, and in nephritis with œdema. It is no established that œdema in nephritis is not due to retention o either sodium chloride or water through any inability of th kidney to excrete these substances. The absolute quantity o water in the blood is not altered in nephritis with œdema; i other words, the blood does not share in the œdema, and sinc water does not accumulate in excess in the circulating blood, th kidney cannot be held responsible for the abnormal retention o water. Similarly, there is no retention of sodium ions in the bloo in nephritis with œdema, but rather the reverse, since the tot serum base, including sodium, is usually reduced. This reductio in serum base cannot be explained by excessive loss of base in th urine, and must therefore be due to an exaggerated tendency fo water and corresponding amounts of salt to pass from the bloo stream into the interstitial spaces. The explanation of this phenomenon still awaits solution, but a major factor is probably th reduction in the plasma protein level. If the water intake i normal, the extent of œdema formation in a potentially œdematou nephritic patient is largely proportional to the salt content of th

diet. Hence restriction of the salt intake by limiting the amount of salt that can migrate to the interstitial spaces along with water is an effective measure in combating œdema. The subject of œdema in nephritis is dealt with in greater detail in Chapter XIV.

(2) It must be administered in conditions where there has been excessive loss of body fluids containing chloride, with resultant hypochloræmia. Among these conditions may be mentioned intense diuresis as in diabetic acidosis, excessive vomiting (*e.g.* pyloric stenosis or upper intestinal obstruction), diarrhœa (enteritis of infants, cholera), or sweating (*e.g.* stoker's or miner's cramp). In hypochloræmia due to any of these causes, the body is in immediate need of fluid and sodium chloride to relieve dehydration and make good the loss of chloride. If they can retain it, patients may be given half-normal physiological saline (½ teaspoonful of salt to 1 pint of water) to drink, otherwise normal saline must be given parenterally.

(3) It must be administered to patients with Addison's disease. In this condition there is a deficiency in the secretion of the cortex of the adrenal glands. As was stated above, this secretion controls the fluid and salt content of the body fluids, and in its absence there is an abnormally large excretion of sodium chloride by the kidneys. Patients with Addison's disease should, therefore, be given up to 10 to 15 grams of sodium chloride daily in addition to that of the diet, in an endeavour to make good this drain. The salt can be given in formalized gelatin capsules, or added to beef tea, soup, etc. In mild cases, this may suffice to keep the patients in relatively good condition, but more severe cases also require injections of adrenal cortical extracts or the subcutaneous implantation of pellets of desoxycorticosterone acetate. Recent work has shown that the requirements of salt and cortical extract are reduced if a diet low in potassium is given. This subject is more of theoretical than practical significance, since experience has shown that of the patients with Addison's disease who cannot maintain health on a normal diet supplemented with sodium chloride, only a small proportion will respond to a reduction of the potassium content of the diet. The great majority of these patients will require hormone therapy, and it therefore seems to us unnecessary to submit such patients to the trouble and difficulty of preparing a diet low in potassium in the limited expectation that this procedure may obviate the necessity of hormone therapy.

Apart from the restriction of sodium chloride intake in cases with œdema, any serious deprivation of salt in the diet is to l deprecated, as it may lead to a marked deterioration of healt Salt-poor diets were introduced in the treatment of migraine an epilepsy, but they have been largely abandoned as little or success followed their use.

Diets containing as little as 0·5 gram of sodium chloride hav been suggested in the treatment of arterial hypertension, but t fall in blood pressure produced is probably due to the loss appetite and consequent under-nutrition caused by such unpala able diets, and salt restriction to this extent seems to be of high questionable value. In fact, there is no indication for rig limitation of salt intake in uncomplicated hypertension. It shou be practised, of course, if congestive cardiac failure develops.

Restriction of sodium chloride intake is one of the principl of the Gerson-Hermannsdorfer diet for the treatment of tube culosis, especially lupus and bone infections. The diet used adequate in protein, rich in fat and vitamins, but restricted i carbohydrate, and no sodium chloride is added to the food. I this country, the same degree of success as was claimed for t diet by the German workers has not been achieved.

Our investigations of chloride metabolism in patients wit marked hypochloræmia have shown that, although a coincide fall in the concentration of salt in the blood, urine and cerebr spinal fluid occurs, the secretion of hydrochloric acid in t stomach remains unaltered. Any attempt, therefore, to influenc the gastric secretion in cases of peptic ulcer or hyperchlorhydri by a rigid restriction of the salt intake is doomed to failure.

CHAPTER VI

WATER IN NUTRITION

All the processes of the body are carried out in a fluid medium, and water is therefore of vital importance in nutrition, being second only to oxygen among the essentials for life.

In normal persons in ordinary circumstances no thought need be given to the water intake, as this is adjusted to requirements by the sensation of thirst. This mechanism is not perfect, however, even in normal persons, as is shown by the loss of weight occurring in many people during hot weather, which is due to failure of the water intake to keep pace with the increased loss of fluid from the skin. It is probably true that under modern civilized conditions some people do not drink the optimum amount of fluid daily.

Under certain conditions, the water balance must be taken into account in the prevention and treatment of disease. In normal persons exposed to high environmental temperatures the increased loss of fluid in sweating necessitates close attention to water intake and urinary output if the deleterious effects of heat on the body are to be reduced to a minimum. Disturbances of the water balance in disease may lead either to an accumulation of fluid in the body – œdema, discussed in detail in Chapter XIV – or to dehydration, which may be due to excessive loss of fluid from the body or to inability of the patient to meet his fluid requirements voluntarily, as in coma or after operations where oral feeding is temporarily contra-indicated.

Water Balance

Intake. It is necessary to differentiate clearly between the total fluid intake, and the intake of fluid as such. The body obtains water from three sources:

1. Ingested fluids.
2. Water contained in solid foods.
3. Water produced in the oxidation of foodstuffs in the body.

The sum of these three constitutes the total fluid intake, and in ordinary circumstances the body actually receives about as much water from the last two together as from the first. The so-called 'dry food' of an average mixed diet contains abou 60 per cent of its weight as water. Such a diet, containing sa 90 grams of protein, 330 grams of carbohydrate, and 150 gram of fat, weighs about 1,500 grams, and contains, therefor 1,500 × 60 per cent = 900 grams or c.c. of water. The wat formed by the oxidation of a gram of protein or carbohydrat is about 0·5 c.c. and that formed by oxidation of 1 gram of f 1·0 c.c. It can be calculated therefore that the diet mentione above as an example would yield about 360 c.c. of water o oxidation in the body. The total water yielded to the body b the so-called 'dry' foods of the specimen diet would therefore b 900 + 360 = 1,260 c.c. This represents about 80 per cent of th weight of the food, so that in calculating an individual's *tot* fluid intake, it would be necessary to add 80 per cent of the weig of the solid food to the amount of fluid taken as such. The norm adult requires about 2,500 to 3,000 c.c. (4 to 5 pints) of wat daily. About half of this requirement therefore is satisfied by th water derived from the solid food, leaving the other 1,200 1,800 c.c. (2 to 3 pints) to be met by water drunk as such, or various beverages.

The Body Fluids

The water of the body may be considered as divided betwe three compartments – the blood, the interstitial fluid, and th intracellular fluid. The total water content of the body compris about 70 per cent of the body weight in an adult, the blo plasma constituting about 5 per cent of the body weight, th interstitial fluid 15 per cent, and the intracellular fluid 50 p cent. The volume of the interstitial fluid is less constant th that of the plasma and the intracellular fluid. It undergoes chang in various physiological and pathological conditions which lea the plasma and intracellular fluid unaltered in volume, the inte stitial fluid thus taking the initial shock of any disturbance in th water equilibrium of the body.

Output. Water excretion occurs through four channels:

(1) *The Lungs.* Expired air is saturated with water vapo and about 300 to 400 c.c. of water are given off through th lungs in 24 hours, the amount depending on the temperature a humidity of the external air and on the level of activity of subject.

(2) *The Intestines.* Except in diarrhœa, only a little water lost in the fæces – about 100 to 200 c.c. daily.

(3) *The Skin.* Water is lost from the skin in two ways: by the ctivity of the sweat glands in secreting sweat, and by the passive iffusion of water through the epidermis – the so-called 'insensible erspiration'. The latter is a purely physical process, and its te, which is roughly parallel to the resting metabolism, shows nly very limited variations. Under ordinary conditions about 00 to 800 c.c. of water are lost by 'insensible perspiration' in 4 hours. In a person leading a sedentary existence in a temerate climate the insensible perspiration is of more importance water loss than the sweat secreted by the sweat glands, but hen hard work is done or the environmental temperature is at r above body temperature so that evaporation of water from e skin and the lungs is the only means of heat loss to the body, arked sweating occurs, and considerable quantities of water of e order of anything up to 15 litres or even more may be lost the sweat daily.

(4) *The Kidneys.* The water that has to be disposed of by the ody after the requirements of the lungs, skin and intestines ave been satisfied is excreted by the kidneys. The kidneys aintain the water balance of the body by excreting any excess f water taken into the body or by reducing greatly the volume f urine excreted if conditions tending to lessen the water content f the body are present, so permitting the requirements of the ther excretory channels to be met.

The capacity of the kidneys to reduce the excretion of urine nnot, however, be pushed to extreme limits without endangering ealth. The excretion of about 500 c.c. of urine per day is essential f all the solids reaching the kidneys for disposal are to be got d of, although this minimum requirement of urine excretion will e less if a diet low in salt is being taken or if large amounts of lt are being lost in the sweat. If the disturbance of water alance is so great that fluid cannot be obtained by the kidneys en for this minimum excretion of urine, waste products of etabolism will perforce accumulate in the body fluids.

Measurement of the Water Balance. From what has been said ove it is clear that the accurate assessment of water balance a matter of considerable difficulty, since it obviously consists f much more than the mere measuring of fluid intake and urinary xcretion. For clinical purposes, however, the measurement of e total fluid intake and output is far too complicated. Actually, nder normal conditions the intake of fluids as such usually alances approximately the urinary excretion. A glance at the figures quoted above will explain why this is so. We saw that t body received about 1,300 c.c. of water from the 'dry' food of ordinary diet. If one adds together the water lost from the bo through channels other than the kidneys, *i.e.* the lungs, skin a intestines, the sum of 1,100 to 1,400 c.c. is obtained, so that tl loss balances approximately the water intake derived from sol food, leaving the kidneys to excrete the water contained in t fluids drunk as such.

Disturbances of Water Balance

The two pathological conditions associated with disturbance of t water balance are retention of fluid in the body (œdema) a a reduction in the water content of the body (dehydration).

1. *Œdema.* As explained in detail in Chapter XIV, œdema not due to excessive intake of water or to any failure in the pow of the kidneys to excrete water, but rather to pre-renal deviatic of water from the blood to the interstitial fluid under the influen of one of three main factors – increased permeability of tl capillary walls, a fall in the colloid (protein) osmotic pressu of the blood plasma, or increased hydrostatic pressure within tl capillaries. It may be pointed out here, however, that throug the capacity of the interstitial fluid to take up the retained wate the plasma volume, and presumably also that of the intracellul fluid, remain practically unaltered in œdema.

2. *Dehydration.* Deprivation of water, either through an inad quate intake or an excessive loss through the kidneys, alimentar tract or skin leads first to a diminution in the volume of th interstitial fluid, and then of the blood plasma but withou alteration of their composition. In the early stages of dehydra tion there is no impairment of blood flow through the kidney and no reduction in the formation of glomerular filtrate; owin to the concentrating power of the kidney, water is conserve without impairing the excretion of solids by the secretion a more hypertonic urine. If dehydration continues, howeve the volume of interstitial fluid and later of plasma become pr gressively smaller, and hæmoconcentration develops, demon strable by an increase in the red cell count and the plasma protei level. The decrease in plasma volume eventually reaches a poin where it causes a diminution in the renal blood flow and henc an impairment of renal function. Due to renal failure and th increased breakdown of body protein that occurs as a result dehydration, there is an accumulation of nitrogenous end-product

of metabolism in the blood. As hæmoconcentration increases and the blood pressure falls owing to lessened blood volume, the hydrostatic pressure becomes inadequate to overcome the osmotic pressure of the plasma proteins, and the formation of glomerular filtrate and hence of urine ceases.

Practical Applications

As has been pointed out above, attention must be paid to the water balance in the following conditions:

1. *Exposure to High Environmental Temperature.* The increased sweating that is essential for the dissipation of heat from the body at high environmental temperatures makes demands on water the magnitude of which is sometimes not appreciated. Persons leading only a moderately active existence in the tropics may lose 6 to 8 litres (about 1¼ to 1½ gallons) of water daily in the sweat alone, and if hard physical work is being undertaken, anything up to about double these quantities may be lost. Accompanying this loss of water there is of course the loss of large quantities of sodium chloride in the sweat, which has been discussed in detail in the preceding chapter, and the combined effect of the marked loss of water and of salt is to make regulation of the water content of the body a difficult matter at high environmental temperatures unless special attention is given to the task.

As we have seen, thirst is not always an accurate guide to the need for water, and the amount of water drunk voluntarily by men working in the heat is often less than that needed to replace the water lost in the sweat. It is very important therefore that persons living and working in the heat should have continual access to plenty of drinking water and that they should be encouraged to drink freely all the water they want, and even more than is required to quench thirst. Contrary to the dangerous and all too prevalent fallacy that persons working in the heat should refrain from drinking water while at work, it has been proved by experiment that the efficiency of men doing hard work in the heat is markedly increased if they are encouraged to drink sufficient water while at work to replace the amount lost in the sweat. An indication of whether persons exposed to high environmental temperatures are drinking sufficient water is afforded by measurement of the urine passed in a 24-hour period – this should never be less and preferably should be more than the 500 c.c. required for excretion of the solids of the urine. Care must be taken, by the measures prescribed in the preceding chapter, th sufficient salt is ingested along with the water drunk.

Deprivation of water from persons working in the heat w lead to a progressive loss of efficiency and the appearance, oft in a few hours, of a type of heat exhaustion specifically caused dehydration and marked by symptoms such as dizziness, las tude, breathlessness, intense sleepiness, and ultimately collaps These symptoms are rapidly alleviated by drinking plain wate

2. *In Cardiac Failure and Renal Disease with Œdema.* In the conditions water passes into the tissues instead of being broug in the blood to the kidneys for excretion. Until the pathologic condition causing this pre-renal deviation of water is relieve the intake of fluid as such must be restricted to an amou approximating to the urinary output, otherwise the œdema w increase.

3. *In Pathological Conditions leading to Dehydration.* Exampl are vomiting, diarrhœa, alkalosis, diabetes, febrile diseases, where fluid cannot be taken by mouth as in coma or followi certain operations on the alimentary tract. In conditions such these an adequate water intake must be ensured, and the patie must be watched for signs of dehydration (sunken facies, d tongue, hæmoconcentration, rising blood urea, etc.).

The best criteria of an adequate water supply are:

(*a*) the volume of the urine – this should not fall below 1,000 c. in 24 hours, especially in febrile conditions where there excessive toxic destruction of body protein.

(*b*) the specific gravity of the 24-hour urine – this should n exceed 1,020.

(*c*) normal elasticity of the skin, a moist tongue and absen of uncomfortable subjective sensations of thirst.

When the patient's condition is such that he cannot ta fluids by mouth, these must be administered parenterally by o or other of the methods described in Chapter XX, the intraveno route being the one usually employed. In most of the conditio mentioned above, loss of salts contained in body fluids (vomitu bowel discharges, sweat, or the urine in diabetes) accompani loss of water, and normal saline is therefore the fluid usual administered parenterally in order to restore the depleted sa and water content of the body. But salt depletion is not invariable concomitant of dehydration; for instance, it will n occur in the post-operative patient who is unable to take anythi by mouth, unless in the presence of a complication such vomiting. It is best not to treat dehydration unaccompanied by salt depletion with normal saline, since this increases the amount of solids and hence of water that must be excreted by the kidneys to maintain the normal osmotic relations of the body fluids. According to McCance and Young (1944), it is a safe rule never to give normal saline unless specifically to replace a loss of salts through vomiting, diarrhœa, diuresis or excessive sweating. All other fluid given should be salt-free or contain much less salt than normal saline. Thus, 5 per cent glucose or 5 per cent glucose in 0·2 per cent saline are suitable solutions for administration by the intravenous route.

It is impossible to state categorically the amount of fluid that should be administered, as this will vary with the degree of dehydration present. The water requirement of a patient who is not dehydrated but is unable to take fluid by mouth will never be less than 2,000 c.c. daily and is more likely to be 3,000 c.c. or even more. If dehydration is present, the fluid intake (fluid administered intravenously plus any taken by mouth) must exceed the output (urine volume, plus 1,100–1,400 c.c. for the water lost via the lungs, skin, and fæces) until dehydration is elieved. The total fluid intake in dehydrated patients should be nything from 3,000–4,000 c.c. a day.

It should be emphasized that dehydration is a very common nd often a very serious complication of disease processes, and he failure to recognize its presence and to correct it is still too requent in medical practice.

THE ACCEPTABILITY OF DIETS

In prescribing a diet to fulfil the fundamental requiremen described in the previous chapters, it must not be forgotten th the average person is less interested in the nutritive value of h diet than in its palatability, satiety value, digestibility, and cos A well-balanced diet must therefore be considered in relation t these factors, as well as those already stressed in connection wit the caloric, protein, mineral and vitamin requirements. For a invaluable review (to which we are much indebted) of the problen of satiety value and digestibility, and of the considerable literatu dealing with these subjects, the reader is referred to McLest (1939).

1. Satiety Value of the Diet

When the stomach is empty, rhythmic contractions (the so-calle 'hunger contractions') occur, which give rise to the sensatio interpreted as hunger, whereas a stomach full of food and physi logically active gives rise to the opposite feeling, a sense of sati faction. Mere distension of the stomach does not promote th sense – it is present only when the stomach and small intestin contain food and when normal muscular and secretory activity going on. The foods which give this sensation of satiety in th highest degree are, therefore, those which remain longest in th stomach and those which call forth the greatest secretion gastric juice. It is on the basis of these two criteria that physi logists assess the satiety value of any particular article of diet.

Meat has a greater satiety value than any other food, and th value is directly proportional to the amount eaten, probabl because the chemical stimulus to gastric secretion provided b the meat extractives is added to the psychic stimulation provide when any palatable food is included in the meal.

Milk and eggs have a relatively high satiety value, but that bread, potatoes and green vegetables is low.

Fat retards the emptying of the stomach, but inhibits gastri secretion. When fat is included in a meal, the total effect of thes two contrary actions is slightly to increase the satiety value.

Sugary foods only increase satiety value if added to a mea meal.

Civilized man has instinctively chosen the type of meal giving the highest satiety value – first, a soup with meat extractives to stimulate gastric secretion, secondly, a meat course with a high satiety value, and finally a more or less sugary dessert, which adds to the satiety value of the meat course.

2. Digestibility of the Diet

To the nutritional scientist, the digestibility of a foodstuff means the percentages of its constituents which can be absorbed by the body. In an average mixed diet, these percentages are 92 per cent of the protein, 95 per cent of the fat and 97 per cent of the carbohydrate. Used in a more popular sense, the term digestibility refers to the absence of discomfort during the process of assimilation. Digestibility in this second sense depends chiefly on the secretory and motor responses which the food evokes in the digestive tract, and these in turn depend on its appeal to the appetite, as well as on the physical state of the food and its chemical composition.

The impressions that have been formed regarding the digestibility of certain foods often spring from a misuse of the food in question rather than from any lack of digestibility. For instance, cheese is often looked upon as being indigestible, but this is because it is usually eaten at the end of a meal or because its digestibility has been impaired by cooking and seasoning. If cheese is taken as part of the protein allowance and eaten during the meal, it is usually well and easily absorbed by the digestive tract.

Rich sugary foods in excess give rise to concentrated solutions of sugar in the stomach, which irritate the gastric mucosa, and may interfere with motor and secretory activity. Taken at the end of a meal, however, the sugary foods are diluted by the previously eaten food and gastric juice, and do no harm, while they increase the satiety value of the meal.

Fat is readily and comfortably assimilated by the digestive tract if taken in moderation. Fried foods soaked in grease are, however, indigestible, because the coating of fat round the food particles prevents the digestive juices acting on them and because frying converts some of the fat into unsaturated compounds (*e.g.* acrolein) which irritate the gastric mucosa. ‘Deep fat’ frying is the method of choice where food is to be fried, because it produces a rapid formation of crust on the surface which prevents the penetration of fat into the interior.

3. Cost of the Diet

Dietary surveys have shown that the less the income availab for the purchase of food, the more do people subsist on a di consisting largely of cereal products. Cereals have the advanta of supplying abundant carbohydrate and Calories at a very lo cost, and they also yield a moderate amount of protein, althou this is of low biological value. A diet consisting largely of cere may supply sufficient Calories, and even sufficient protein, but can be criticized on three grounds: (1) that the protein is of lo biological value; (2) that it contains insufficient calcium and iro and (3) that the vitamin content is inadequate.

The diets of the two lower income groups in Table 5 (p. illustrate the effect of an excess of cereals on the composition the diet. In Group 2, in which the diet is adequate in prote and Calories, it will be seen that the mineral and vitamin requir ments are not met. Also, only 42 per cent instead of at lea 50 per cent of the protein intake is in the form of animal protei The addition of a liberal amount of milk and milk produc (*e.g.* cheese) makes a cereal diet adequate except for iron a vitamin C.

The food budgets of the richer classes, on the other han usually contain too large a proportion of meat and not enoug fresh fruit and vegetables. Such diets, although rich in prote of high biological value, can be criticized on the ground that rich meat diet is relatively poor in vitamins and calcium.

Therefore, when economy must be practised, too much of th protein should not be taken as meat, as this means that too litt is spent on fruit and vegetables. An adequate diet can be secure with the lowest expenditure on protein by combining milk an milk products with cereals, legumes and herrings.

For those of moderate means, the following rules were suggest by Sherman (quoted by McLester, 1939) to secure an efficie diet at an economical cost. (1) At least as much should be spe on milk (including cream and cheese, if used) as on meats, poult and fish; and (2) at least as much should be spent on fruits an vegetables as on meats, poultry and fish.

It is worth recalling in connexion with these rules that herrin are the cheapest source of the valuable combination of first-cla protein, fat, vitamins A and D, and iodine.

CHAPTER VIII

THE COMPOSITION AND CHARACTER OF FOODS

THE value of the different types of foodstuffs in meeting the various nutritional requirements set forth in the preceding chapters has already been mentioned when dealing in detail with these requirements. The subject is approached from another angle in this chapter by summarizing the advantages and the defects of the various classes of food products.

DAIRY PRODUCTS

1. Milk

Milk and eggs are the only two foods designed by Nature to form the sole nourishment of the developing young of the higher forms of animal life, and milk is hence often spoken of as a complete food. This statement needs qualification, however, even in the case of the young infant whose only food is milk, since modern dietetic practice demands that supplements of vitamins C and D should be administered. Milk contains the main constituents of an adequate diet (*i.e.* protein, fat, carbohydrate, ample calcium and phosphorus and varying amounts of the vitamins essential to man), but these nutrients are in dilute solution since milk is seven-eighths water, and certain essential nutrients, such as iron, aneurin, nicotinic acid and ascorbic acid, are present in only small amounts.

The Proteins (caseinogen, lactalbumin and lactoglobulin) are of the highest biological value, supplying all the animo-acids necessary for health. Milk is therefore a valuable supplement to a cereal diet.

The Carbohydrate – lactose – is broken down in the intestine to glucose and galactose. In the bacterial fermentation of milk, the lactose is broken down to lactic acid. The use of milk fermented by cultures of *Lactobacillus acidophilus*, a normal inhabitant of the intestinal tract, has been in vogue for a number of years in the treatment of intestinal dyspepsias, the purpose being to change the nature of the intestinal flora and bring about disappearance of putrefactive organisms. The therapeutic value of such treatment is not universally accepted. Soured milk is, however, mo digestible than ordinary milk, since the curd formed in t stomach is finer, and the lactic acid neutralizes some of th buffer substances in cow's milk which interfere with gastr digestion. For this reason, milk soured with lactic acid is no often used in the artificial feeding of infants.

The Fat is present as an emulsion of extremely fine drople the small size of which renders milk fat easily digestible.

The Salts. The mineral content – chiefly calcium, phosphoru potassium, and sodium – is high; indeed milk and its produ cheese are the only natural foods of animal origin that are ri sources of calcium. Since this mineral is present in small amour in food of plant origin, except in some of the leaf vegetabl milk and cheese occupy a unique position in the diet as sourc of calcium. Milk has a low iron content, but such iron as is prese is entirely in the available form. There is a danger that too lit iron may be given to an artificially fed infant when the co milk is diluted to reduce its protein content.

The Vitamins. Milk is a useful source of vitamin A. The actu content depends largely on the diet of the cow, summer m having a much higher vitamin A content than winter milk. value of 30 International Units of vitamin A per ounce of m can be accepted as an average figure. It also contains B_1 (4 Int national Units per ounce) and riboflavin and C (0·3 mg. of ascor acid per ounce) in appreciable amounts, but vitamin D is prese only in small and variable amounts (0·1 to 1·0 Internatio Unit per ounce). Pasteurization of milk has little or no eff on its content of A, B_1, riboflavin and D, but it does destr part of the vitamin C though less than is usually believed, o about one-fifth of the vitamin C being lost in the process. T boiling of milk causes a greater fall in vitamin C content th pasteurization. Any loss of vitamin C caused by pasteurizat is more than made good by the supplement of orange juice wh every artificially fed infant should receive (*i.e.* two teaspoonf daily up to the age of four months, thereafter one tablespoon daily). In view of the value of pasteurization in preventing mi borne infection, the idea widely held that pasteurization low the nutritive value of milk must be challenged. There is evidence that pasteurization reduces the nutritive value of m in any other way than by lowering its already low vitami content, and this is easily remedied by the orange juice supp ment mentioned above.

Exposure of milk to bright light causes rapid destruction of its riboflavin and slow destruction of its vitamin C.

The nutritional value of milk is therefore due to the high biological value of its proteins, the digestibility of its fat and the contribution of calcium, phosphorus, vitamin A and riboflavin made to the diet in the amounts usually consumed. The large-scale experiments mentioned in Chapter I have shown convincingly the improvements in growth, general nutrition, and the condition of the teeth which result from providing children with an adequate amount of milk. One pint of milk daily should be taken by an adult, and 1½ to 2 pints daily by a growing child. It should be noted that milk is an economical as well as a valuable food.

Milk has additional advantages over other sources of first-class protein. Meat and fish have an acid ash whereas that of milk is alkaline, so that milk assists in maintaining the alkali reserve of the body. Milk also appears to have the property of maintaining the normal intestinal flora, thus favouring synthesis of vitamins of the B group. For example, although it contains very little nicotinic acid, milk is of use in the dietary treatment of pellagra, possibly because it promotes the formation of nicotinic acid by the intestinal bacteria.

Skimmed Milk. In this and other countries before the war, most of the skimmed milk left after separation of cream was ither fed to animals or even discarded, there being little demand or it as human food. This was due chiefly to prejudice, based n the erroneous conception that skimming removes most of the nutritive value of milk.

Skimming removes practically all the milk fat, and with it the t-soluble vitamins. But the valuable milk proteins, the calcium nd phosphorus, and the water-soluble vitamins, of which ribo-avin is the most important in milk, are left. Accordingly skimmed lk, far from being of little nutritive value, is on the contrary valuable source of essential nutrients. Milk fat has no funda-ental nutritional advantage for adults over other forms of fat cept for its vitamin A content, and this vitamin can of course supplied from other sources. These remarks do not apply to fants, in whose case the ease of digestion of milk fat is a very portant dietetic factor. The lack of fat, and hence of energy lue, in skimmed milk therefore renders it an unsuitable food infants.

The allowance of milk recommended by most authorities –

1 pint per day for adults, and up to 2 pints per day for childre and adolescents and pregnant women – may be beyond the mean of the lower income groups. Ideally, of course, this situatio should be met by a reduction in the price of whole milk to level that would make its purchase in the required amount possible to all. But until that happy state of affairs can b achieved, an increase in the amount of skimmed milk markete for human consumption would help to ensure that everyone coul obtain the recommended daily allowance. Liquid skimmed mil admittedly is not so pleasing to the palate as whole milk whe consumed as such, but it can be used in many cooking procedures just as well as whole milk (*e.g.* in the making of custard and sauces and in baking) and with no loss of palatability in th products. In the dried form, too, skimmed milk can be incorporated in cooked foods like bread, cakes and puddings in greate amounts than liquid milk, and the nutritive value of these food is thereby enhanced. War-time experience in the use of drie skimmed milk should have done much to break down the prejudic against its domestic use. It would be of great advantage, especially to people in the low income groups, if the sale of drie skimmed milk were made a permanent part of post-war nationa nutritional policy. Skimmed milk prepared by the spray-dryin process, where the milk is forced through a fine jet into a stea heated metal drying chamber in which the small milk droplet are dried almost instantaneously, is more palatable and mor easily reconstituted than milk dried by the roller process, i which the milk is allowed to flow in a thin film over revolvin metal steam-heated rollers, from which the film of dried milk scraped by fixed metal blades.

From the point of view of food economics, too, the use skimmed milk as human food instead of as animal food has muc to recommend it. Thus, its nutrients are at least equal, poun for pound, to those of muscle meat when used as human foo whereas it has been calculated that it needs 10 lb. of the nutrien of skimmed milk to produce 1 lb. of food nutrients in the for of pork.

Milk Products

1. *Butter* is formed by the coalescence of the fat droplets soured milk during churning. Good butter contains about 90 p cent of fat and 8 per cent of water, the remaining 2 per ce consisting of minerals, casein and lactose. Butter provides fat

an easily digested form, and it is of importance in nutrition because of its high fuel value, and also because it contains the fat-soluble vitamins of milk. It is, therefore, a rich source of vitamin A, containing on an average about 1,100 International Units per ounce, the vitamin A content being lowest in winter and highest in summer. The vitamin D content is 5 to 30 International Units per ounce.

Butter Substitutes. Butter is for many people an expensive article of diet, and for this reason comparatively inexpensive substitutes have been introduced, consisting of mixtures of various fats blended and treated so as to form food products which resemble butter in flavour, colour and consistence. Such food products, known under the general name of 'margarine', are manufactured from emulsions of milk with vegetable or animal fats. Margarine forms an effective alternative to butter as regards caloric value, but is lacking in vitamins, unless these are specially introduced. The isolation and preparation of vitamins on a commercial scale resulted in a number of excellent brands of 'vitaminized' margarine being put on the market as far back as 1927, when concentrates of vitamins A and D were effectively and uniformly incorporated. It may be noted that since February 5th, 1940, the introduction of vitamins A and D into all brands of margarine offered to the public for table use has been made compulsory. The stipulated amounts of these vitamins in margarine are at the present time 570 International Units of vitamin A nd 56 International Units of vitamin D per ounce, so that margarine contains rather more vitamin A than winter butter nd considerably more vitamin D than even summer butter.

2. *Cheese* is essentially the curd formed by the action of rennin n the caseinogen of milk. Cheese of the Cheddar type contains bout 25 per cent of protein, 34 per cent of fat, 6 per cent of actose and minerals and 35 per cent of water. Being rich in rotein of high biological value and in easily assimilable fat, heese is highly nutritious. It also contains most of the calcium, hosphorus, iron and vitamins of milk. Since much of the milk t is present, cheese is a rich source of vitamin A, containing ughly 400 International Units per ounce. It is also a rich source riboflavin. To obtain its full value, cheese should be given its roper place in the dietary (see p. 167), and not be used as an nnecessary supplement to an already adequate diet.

2. Eggs

Since the egg forms a complete food for the embryo chick, must therefore be rich in essential nutritive substances.

The proteins are of high biological value, and the fat, which all in the yolk, is finely emulsified, and hence easily assimilate An average egg contains roughly 6 grams each of protein a fat, and yields about 80 Calories.

The minerals and vitamins exist mainly in the yolk, which also a valuable source of calcium, phosphorus and iron. The yo is a rich source of vitamin A (about 2,000 International Units p ounce); and vitamin D (about 100 International Units per ounc vitamin B_1 (about 30 International Units per ounce), and rib flavin are also present. The white of egg is one of the best sourc of riboflavin.

Eggs have, therefore, a high nutritive value, which is especia useful for growing children and for adults recuperating from illness.

MEAT

Under this heading are discussed the organs, as well as mus meat, poultry and game. Meat contains approximately 20 p cent of protein, 70 per cent of water and variable amounts fat and extractives.

Muscle Meat

The Proteins of meat are of high biological value, and it is d to its richness in good protein that meat finds an import place in the dietary.

The Fat is present in variable amounts. In beef, it is mai in layers, separable from the lean, but in pork and duck, the is also deposited in minute particles throughout the muscle well, which tends to make them less digestible. The variation the fat content of different cuts of meat is of significance w the caloric value of a diet has to be adjusted accurately. The content of meat varies from 4 grams of fat per ounce of l meat, when all visible fat has been removed, to 12 grams ounce (including visible fat) in mutton chops.

The Extractives. These consist chiefly of creatine, purine ba and glycogen. Meat extracts and beef tea consist chiefly of th extractives, and until recently it was considered that s beverages, although pleasant to take, had little food val

Vhile this is probably true in the case of beef tea, recent advances n methods of the estimation of vitamins in foodstuffs have evealed that concentrated meat extracts are rich sources of iboflavin and nicotinic acid and probably also of the other nembers of the vitamin B_2 complex present in meat. Beef tea nd meat extracts are of use in dietetics on account of their mild timulant action on the secretion of gastric juice, but meat extracts nust also be regarded as a reasonably good source of vitamins f the B_2 complex.

The Vitamins. Muscle meats in the amounts normally consumed nake an important contribution to the dietary intake of certain f the B group of vitamins. Meat is not a rich source of aneurin, lthough lean pork is an exception to this statement, the richer neurin content of pork flesh probably being due to the higher roportion of aneurin in the food given to pigs than in the diet f other meat-producing animals. But meat contains relatively rge amounts of riboflavin and nicotinic acid and probably of e other vitamins of the B_2 complex. It has been estimated at the amount of meat in the normal British or American etary supplies about half the total nicotinic acid and one-arter of the total riboflavin content of the diet.

Muscle meat contains a little vitamin A in the associated fat, t very little ascorbic acid. What little vitamin C is contained meat, along with the somewhat larger amounts present in gans such as liver, is, however, sufficient to protect from scurvy oples like the Eskimos whose diet is almost entirely confined meat. In practical dietetics, however, it is usual to consider eat, as containing no vitamin C.

Cooking causes a loss of vitamin B_1 from meat, although some this loss is prevented by cooking methods which retain the at juices, *e.g.* frying. In canning, the loss of vitamin B_1 is eater and may be as high as 70 to 80 per cent. There is a gressive loss of the remaining vitamin B_1 on storage of canned t, especially if this takes place at high temperatures.

he Minerals. Meat is rich in phosphorus, but very poor in cium. Of their high total iron content almost 100 per cent of iron of the organs is available to the body compared to only to 25 per cent of the iron of muscle meat.

rozen and Chilled Meat. Frozen meat has been shown to ain free from bacteria for over a year. Beyond some surface ing, it undergoes little appreciable physical, and no chemical, nge, and in its nutritive value and wholesomeness it is equal

to fresh meat. When meat is merely chilled, that is, kept at
temperature of about 2° C. (*i.e.* just above freezing point
bacteria can be detected in it after a month, and it has no
therefore, the keeping qualities of frozen meat.

The Organs

The same remarks apply to the organs as to muscle meats, exce
that the proteins are of even higher biological value and t
vitamin content is much higher. Thus, liver, heart and kidn
all contain much larger amounts of aneurin, riboflavin and nic
tinic acid than muscle meat. Liver, in addition, is the riche
animal source of vitamin A among the common foodstuffs,
liver containing over 4,000 International Units per ounce a
sheep's liver over 12,000 International Units per ounce.

In practical dietetics it must be remembered that the supp
of organs is limited – for instance, liver constitutes only abo
2 to 3 per cent of carcass meat. It follows that liver can nev
form a large part of the available food supply, and the dietitia
duty therefore lies rather in seeing that it is taken by tho
who most need it, such as patients with pernicious anæm
or a deficiency of the vitamin B complex, than in urgi
a general increase in its consumption by the population as
whole.

White and Red Meat

There is one tradition in dietetics that dies hard, and that is t
popular idea that the white meats (poultry and game) are super
to red meats (beef and mutton) for the treatment of the sick
account of their greater digestibility and their lower percent
of nitrogenous extractives, especially purine derivatives. Wh
meat is of looser texture and contains less fat and connect
tissue than red meat, and hence may be more digestible.
there is no difference in the nutritive value of white and
meat as regards protein content, etc. Furthermore, the pur
content of white meat is as high as that of red meat, if not high
It is entirely wrong, therefore, to suppose that a patient wh
protein or purine intake has been restricted for therape
reasons can take white meat with impunity provided that
intake of red meat is kept low.

The advantages and disadvantages of meat from the diet
point of view can be summarized thus:

Advantages. 1. Its flavour and palatability have a stimulat ffect on the appetite, and hence on the psychic secretion of astric juice.

2. Its satiety value is high.

3. It forms a source of protein of high biological value.

Disadvantages. 1. Excessive meat consumption restricts the ntake of other valuable foods.

2. The more expensive cuts of meat are an uneconomical source f protein.

3. Compared with such sources of first-class protein as milk nd cheese, meat is deficient in calcium and vitamins, except for icotinic acid and to a less extent riboflavin.

4. Meat has a relatively high purine content – a fact which ust be borne in mind in the treatment of the gouty diathesis.

If the diet is otherwise normal, one helping of meat a day is ufficient to ensure an adequate protein intake.

FISH

oth in its protein and purine content the flesh of fish resembles ther forms of meat. The protein is as high in biological value, ut, except for such species as salmon, mackerel and herring, the t content is lower. Cheap fish, such as the herring, can provide rotein of high biological value at a low cost and yet in a palatable rm, and the importance of such a source of good protein in the et of the poor cannot be overstressed. The liver oils of many arieties of fish are rich in vitamins A and D. The bodies of fatty h such as herrings, salmon, pilchards and sardines are also od sources of vitamin A and especially of vitamin D.

It has already been mentioned (p. 147) that, apart from drinking ater and vegetables, fish is the best dietetic source of iodine. general, the muscle tissue of fish contains considerably more cium than that of meat; and canned fish, the softened bones which are generally eaten along with the flesh, may contribute latively large amounts of calcium to the diet. Owing to the lative infrequency with which fish appears at table and the ount eaten when it does, fish cannot, however, compare with ilk as a source of calcium.

The digestibility of fish compares well with that of meat.

Shellfish. The types used for food are the molluscs (*e.g.* oysters, ussels) and the crustaceans (crabs, lobsters, shrimps).

The molluscs resemble other forms of meat or fish in composition, except that the glycogen content is higher. They are excellent food, and are easily digested. Their vitamin content higher than that of other forms of fish or meat, and the high iodi content is of value also. The possibility of infection from she fish grown in contaminated water has now been eliminated this country.

The crustaceans resemble the molluscs as regards nutriti value, except that their higher fat content makes them less ea to digest.

The not infrequent occurrence of allergic idiosyncrasy to she fish must not be forgotten.

CEREALS

From the point of view of quantity consumed, the cereals a the most important of the foodstuffs. They provide anythi from 30 per cent to over 75 per cent of the energy value of t diet in different parts of the world. The principal cereal grai used in human dietaries are in descending order of the amou produced and consumed, rice, wheat, maize, rye, barley, mil and oats.

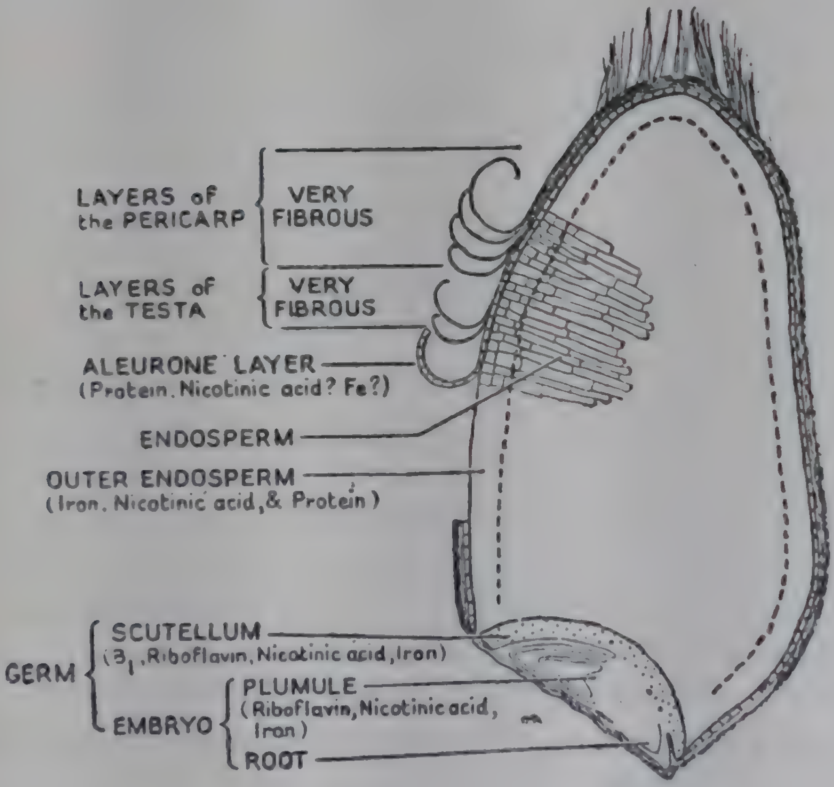
The reasons for the predominance of cereals in the diet of m are:

1. The ease with which they can be produced, stored, a transported.
2. Their cheapness.
3. The ease with which they can be prepared for the table.
4. Their palatability.

The Nutritive Value of Cereals. For a proper understanding the nutritive value of the cereals and of the changes produc therein by the processing to which they are submitted in th preparation as food for man, it is necessary to consider t structure of the cereal grain and the composition of its par The wheat grain will be considered in particular, since it is chie in the case of wheat that the composition of the various pa of the grain has been studied in detail. In general, however, th observations apply also to the other cereal grains. Interest the relative nutritive qualities of flours containing varying p portions of the components of the cereal grain has been intensi in Britain by the measures taken during the second World W to increase the yield of flour from wheat and to enhance utritive value without impairing its palatability. These quesions continue to be of great significance in a world where a eneral shortage of foodstuffs has tended to emphasize the utritional value of cereals in the diet. For these reasons we ropose to consider in some detail the problems involved.

As shown in the diagram, which has been reproduced from a aper by Moran and Drummond (1945), the cereal grain consists f four main parts:

(*a*) *The Germ.* This contains the embryo plant and forms just nder 3 per cent of the whole grain. Separating the embryo plant


LAYERS of the PERICARP
VERY FIBROUS
LAYERS of the TESTA
VERY FIBROUS
ALEURONE LAYER
(Protein. Nicotinic acid? Fe?)
ENDOSPERM
OUTER ENDOSPERM
(Iron. Nicotinic acid, & Protein)
GERM
SCUTELLUM
(B1, Riboflavin, Nicotinic acid, Iron)
EMBRYO
PLUMULE
(Riboflavin, Nicotinic acid, Iron)
ROOT

(*After Moran and Drummond*, 1945)

om the remainder of the grain is a sheath of powdery matter own as the scutellum. The embryo is rich in protein and oil. contains appreciable quantities of riboflavin, nicotinic acid and on, while its oil is the richest known natural source of vitamin E. e scutellum is of particular importance in that, while forming ly about 1·5 per cent of the total bulk of the grain, it contains less than 60 per cent of the total vitamin B_1.

(*b*) *The Endosperm.* This constitutes the food store for the veloping embryo plant, and comprises about 85 per cent of e bulk of the grain. It consists chiefly of starch, but contains me protein and also iron and nicotinic acid. The concentration of these nutrients is not uniform throughout the endosperm, l increases from the centre of the grain outwards towards pericarp, so that the outer layers of the endosperm are richest in iron and nicotinic acid.

(*c*) *The Aleurone Layer*. This is a thin cellular layer surround the endosperm and rich in protein of higher biological va than that of the endosperm.

(*d*) *The Pericarp*. This constitutes the outer fibrous cover of the cereal grain. It is rich in phosphorus, but since this chiefly present as phytic acid, it is largely unavailable to body. The pericarp, with the aleurone layer and adherent partic of the outer more nutritive layers of endosperm, forms the b that is removed in milling.

Yellow colouring matter in a cereal indicates either the prese of carotenoid pigments of which, however, only a small proport is biologically active, or the presence of riboflavin. The o cereal with a content of biologically active carotinoids suffici to make a significant contribution to the vitamin A intake yellow maize.

Before the roller mill process for milling wheat was introdu into Britain in 1877, flour was produced in relatively crude m by grinding the grain between two flattened mill-stones. parts of the grain were incorporated in a wholemeal flour. addition a more refined flour was made by sifting the total prod through muslin which removed some of the fibrous bran lay This refined flour, however, contained the germ, and much the aleurone layer and outer endosperm, and so retained greater part of the nutritive value of the whole cereal grain

Different stages in the roller mill process for the product of 'white flour', as the term is understood to-day, remove f the bran, including the aleurone layer and most of the out more nutritious part of the endosperm, and then the ger including the scutellum. The white flour that is left cons almost entirely of endosperm, containing starch, the endospe proteins, glutenin and gliadin, a little inorganic material, chi potassium, and minimal amounts of vitamins. White flour thus a good source of energy, but is low in proteins, and th it contains are of lower biological value than the proteins carded in the aleurone layer and germ. Its vitamin content very low, and what little riboflavin is present is largely destro by oxidation in the bleaching process to which white flour subjected in order to increase its whiteness.

In their masterly review of the diet in this country throughout e centuries (*The Englishman's Food*, 1939), Drummond and Wilbraham give the following comparative analysis of the nutri- ve value of stone-ground 'white' flour and bleached white flour milled by the roller process:

COMPOSITION PER 100 GRAMS

Nutrient		Stone-ground white flour	Roller-mill bleached white flour
Protein	(gram)	12·5	10·1
Fat	,,	1·4	0·9
Calcium	(mg.)	44·0	20·0
Phosphorus	,,	180·0	92·0
ron	,,	3·3	1·0
Riboflavin	,,	0·02	0·01
Vitamin B_1	I.U.	100·0	10 to 15

The nutritive value of roller-mill 'white' flour is therefore con- derably lower in all respects than that of stone-ground 'white' our. Drummond and Wilbraham point out that the increased tensity of the demand for white bread in England that occurred uring the nineteenth century was doubtless explained in great easure by a reaction on the part of the people against the dulteration of wheat flour by the addition of potato flour, barley, ran and other products that darken flour. This adulteration as frequently resorted to during the years of want accompanying nd following the Napoleonic wars. The introduction of the ller-mill, which enabled the greyish-yellow germ as well as the an to be removed from flour, made it possible for the millers meet this demand of the public for white bread. While the eld of flour was smaller than with stone-grinding, the millers re able to offset this loss by selling the discarded portions he grain (the so-called offals) as foodstuffs for livestock. The ller-mill process had other economic advantages which help to plain its rapid substitution for the older method of milling. ller-mills were found to be quicker in operation and cheaper maintain, and the white flour produced could be stored for a nger time without deterioration than stone-ground white flour wholemeal flour. The enhanced keeping qualities of roller-mill hite flour over wholemeal flour are due to two factors. Firstly, e larvæ of the weevils that infest grain cannot flourish without e valuable nutrients present in the germ of the cereal grain and white flour therefore will not support the life of these lar whereas they can develop in wholemeal flour. Secondly, the of the embryo when exposed to the oxygen of the air tends become rancid, hence spoiling the taste of flour. Wholemeal fl therefore cannot be handled in commerce so readily as wh flour, from which the embryo has been removed, without dan of deterioration.

Drummond and Wilbraham have emphasized that the cha from stone-ground flour to roller-mill flour was the most import cause of the rapid deterioration of the British diet that occur in the closing years of the nineteenth century, and was larg responsible for the grave impairment of physique in the popu tion which became evident following this change of diet. As example of the deterioration of the nutritive value of the d which followed the introduction of the roller-mill, they cite following figures:

Diet	Vitamin B$_1$ – International U
Navy Ration, 1811 . . .	1,050
Prison Diets, 1843 . . .	1,100 to 1,300
Prison Diets, 1864 . . .	1,000 to 1,300
Poverty Diet, 1939 . . .	220

The poor, who were more dependent on bread to supply dietary essentials than those who could better afford to supp ment their diet with other foods, were, of course, most affect While frank deficiency diseases did not become common, m bidity and mortality statistics point to a state of public hea at the turn of the century that would be considered appall to-day. It is impossible to escape from the conclusion t malnutrition was largely responsible for this unhappy state affairs.

Apart from a temporary increase during the first World W in the proportion of the grain included in flour (known as extraction rate of the flour), roller-mill white flour (70 per c extraction) has remained the standard flour in use for more th fifty years. A general improvement in the standard of liv and hence an increased consumption of foodstuffs other th bread has, however, tended to offset the lowered nutritive va of flour except among the very poor whose diet, as has be explained in Chapter I, was still very deficient in many of essential nutrients.

When the second World War broke out, the question of extraction rate of flour became a problem of urgent natio importance for two reasons: first, it was calculated that an increase in the extraction rate of flour, by increasing the yield from imported grain, would save half a million tons of shipping space a year, and secondly, this measure would provide increased amounts of certain essential nutrients – protein, iron and vitamins of the B group – the intake of which was reduced by the system of food rationing that had to be enforced early in the War (see Chapter X).

The maximum saving of shipping space would of course have been secured by the milling of wholemeal flour, but several considerations concerned with nutrition, keeping qualities and palatability militated against such a solution of the problem. It is of importance to consider these matters in some detail.

(a) *Roughage*

The fibrous pericarp of grain cannot be digested by man, and thus the use of wholemeal flour in place of white flour increases the amount of indigestible bowel residue. This is undoubtedly of great benefit to many people, since constipation is extremely prevalent in the so-called civilized races. An excess of roughage n the diet may be undesirable in certain types of gastro-intestinal disease. Since sufferers from such conditions constitute a very small minority of persons, it is difficult to see why the advantages of an adequate supply of roughage should be withheld from the rest of the community.

b) *Digestibility*

t has been shown by several workers that the digestibility of lour (in the scientific sense, *i.e.* the proportion of nutrients of a oodstuff absorbed from the intestine) decreases with an increase n the extraction rate. This decrease in digestibility holds good or protein, carbohydrate and minerals.

As a result of the increased proportion of non-utilizable bran n wholemeal flour and the decreased digestibility of its carbohydrate and protein, it has a lower energy value than white lour. From the point of view of nitrogen balance, however, the owered digestibility of the protein of wholemeal flour is com-ensated for by its higher protein content and the higher bio-ogical value of its proteins as compared with those of white our. Much of the phosphorus of the fibrous parts of the bran s present in the form of phytic acid (inositol-phosphoric acid), which is not digested in the human intestine so that its phosphorus is non-available to the body. This is of little physiologi importance, since phosphorus is present in abundance in ot foods than cereals.

Phytic acid, however, has more serious effects than merely render some of the phosphorus of the diet unavailable to body. It forms very insoluble phytates with both calcium a iron, and by combining with these essential elements it preve their absorption. This effect has been clearly demonstrated human experiments by McCance and Widdowson (1942) w showed that much less calcium was absorbed from a diet in wh wholemeal bread provided about 40 to 50 per cent of the to Calorie intake (*i.e.* about twice the proportion in a normal mi diet) than when a similar amount of white bread was eat although the calcium intake was somewhat greater when diet contained wholemeal bread. They proved, too, that t difference in calcium absorption was due to the phytic a content of the wholemeal bread. A similar depressing action phytic acid on the absorption of iron was found by Widdow and McCance (1942), although the evidence was perhaps not convincing as in the case of calcium. The absorption of iron w significantly lower from the diet referred to above, in wh wholemeal bread replaced white bread, in spite of the fact t the iron intake was about 50 per cent higher. Addition to wholemeal bread of sufficient calcium to combine with the ph acid did not increase the absorption of iron up to the level attai on the white bread diet.

Widdowson and McCance suggest that the depressing eff of wholemeal bread on iron absorption may be due to t increased amount of inorganic phosphate in wholemeal bread. well as to the phytic acid present therein. They concluded t wholemeal cereals cannot be regarded as superior sources of i to milled cereals, even although the former contain more ir Nevertheless, in the final analysis the best method of assess the nutritive value of a food is the study of the clinical effe produced on human beings who consume it. We have fou (Davidson, Donaldson, Lindsay and Roscoe, 1944) that cor sponding with the period during which 85 per cent extract bread was eaten (1942 to 1944) there occurred a steady a significant rise in the hæmoglobin levels of school-children a pregnant women in Edinburgh. Whether this satisfactory res can be attributed entirely to the increased consumption of ir or whether other unknown factors were in part responsible, beside the point, and does not detract from the importance of these observations.

Although a certain amount of enzymatic destruction of phytic acid occurs when bread is baked with yeast, the complete destruction of phytic acid by this means is not a practical proposition on a commercial scale. McCance and his co-workers (1942) accordingly investigated the effect of overcoming the deleterious effect of phytic acid on calcium absorption by adding calcium (in the form of creta preparata, a neutral form of calcium carbonate) to wholemeal bread. They found that the addition of 200 milligrams of calcium carbonate to every 100 grams of wholemeal (92 per cent extraction) flour allowed as much calcium to be absorbed from the diet as when the wholemeal bread was replaced by white bread.

When it became necessary to increase the extraction rate of flour in order to save shipping space, the question arose as to what was the optimal extraction rate which would accomplish this object and at the same time avoid the undesirable effects of 100 per cent extraction flour on persons with digestive disorders and on the absorption of calcium and iron. In 1941 the Medical Research Council issued a memorandum recommending that the extraction rate of flour should be fixed at 85 per cent. The standards laid down were that the flour should contain not less than 1 International Unit of vitamin B_1 per gram, and not more than 0·9 per cent of fibre (as against a fibre content of less than 0·2 per cent in white flour). The experiments of Krebs and Mellanby (1943) had shown that bread made from 85 per cent extraction flour depressed calcium absorption, though not to the same extent as wholemeal bread, and that it required 120 milligrams of calcium per 100 grams of flour to permit the absorption of optimal amounts of calcium. The Medical Research Council therefore recommended that 130 milligrams of calcium should be added to every 100 grams of 85 per cent extraction flour.

In March, 1942, the Government ordered that all flour milled in Britain should be of 85 per cent extraction, and that it should conform to the specifications recommended by the Medical Research Council. On the grounds of technical difficulties in breadmaking, however, the amount of calcium authorized by the Ministry of Food to be added to this so-called National Flour was only 65 milligrams per 100 grams of flour (*i.e.* about half that recommended by the Medical Research Council). According to McCance and Widdowson (1944) this amount of calcium

is about that required to neutralize the phytic acid which esca
destruction by the enzyme phytase present in flour when bre
from 85 per cent extraction flour is baked, so that the fortifi
national bread was approximately equivalent to unfortified wh
bread with regard to calcium absorption.

A glance at Table 17 indicates the marked improvement in t
nutritive value of the 85 per cent extraction flour as compar
with white flour (70 to 72 per cent extraction). Bread baked fr
85 per cent extraction flour contains over three times as mu
vitamin B_1 and riboflavin as white bread, its iron content
more than doubled, and the protein content is somewhat high
The diet of a person consuming 10 oz. of bread per day ma
from 85 per cent extraction flour in place of 10 oz. of white bre
would be enriched by the following amounts: 130 Internatio
Units of vitamin B_1 (*i.e.* almost 25 per cent of the proba
optimum daily requirement), 0·2 milligram of riboflavin (*i.e.* abo
as much as is provided by 5 oz. of milk or $1\frac{1}{2}$ oz. of cheese) a
3 milligrams of iron (*i.e.* almost as much as in 3 oz. of mea
although it is not certain how much of this iron is available
absorption.

The introduction of National Flour in 1942 has led to a co
siderable amount of controversy regarding the advisability
the retention of this measure as a permanent feature of natio
nutritional policy. Much of the argument has centred round t
alleged public demand for white bread when the exigencies
war no longer necessitate the conservation of shipping spa
and the desirability of releasing wheat offals as food for livesto
in order to build up the pig and poultry industry to its pre-w
levels. Those who favour the reintroduction of white flour as t
basic cereal product claim that the public demand for wh
bread can be met and the nutritional advantages of high-extr
tion flour can be achieved by the policy adopted in the Unit
States of fortifying white flour with vitamins (aneurin, rib
flavin and nicotinic acid) and minerals (iron and calcium)
optimal amounts. In parenthesis, it may be pointed out th
vitamin D is also being added to white flour in the United Stat
but this is not relevant to the present discussion, since vitamin
is not a natural constituent of the wheat grain.

The contention that the fortification of white flour makes
as valuable to man nutritionally as high-extraction flour is bas
on the fact that the enrichment policy adopted in the Unit
States does in fact supply even greater amounts of these nutrien

than are present in high-extraction flour. It must be admitted that at the present there is no conclusive evidence that the other known B vitamins in wheat, such as pantothenic acid and pyridoxine, are essential in human nutrition, or that nutritional health would necessarily suffer as a result of the absence from the diet of the amounts present in high-extraction flour. The possibility exists, however, that nutritional factors as yet unknown may be present in high-extraction flour. It would seem therefore more reasonable from the nutritional point of view to ensure the inclusion in the diet of as many as possible of the natural nutrient factors in wheat, just as it seems illogical, in the absence of any evidence that high-extraction flour is not an eminently suitable foodstuff for man, to remove natural nutrients from flour and then replace them by synthetic products.

It must be emphasized here that the gloomy prognostications of a few opponents of the high-extraction flour regarding its harmful effect in disorders of the digestive tract have not been borne out by the experience of the senior author and other physicians specializing in diseases of the alimentary tract. There is no evidence to suggest any increase in the incidence or delay in the healing of ulcerative colitis, peptic ulcer or gastritis consequent on the introduction of National Flour. If anything, the evidence is in the contrary direction, possibly because of improvement in the general health of the patients as a result of consuming bread of high nutritive value. Physicians are in agreement that the incidence of constipation markedly decreased during the period when 85 per cent extraction flour was in use.

We do not wish to enter into the controversy whether a widespread public demand for white bread does in fact exist or whether it has been fostered by certain vested interests for commercial reasons. We would, however, point out that the best way to meet any such demand is for all scientists and medical men who believe in the value of a high-extraction flour to conduct a vigorous and sustained campaign against the ignorant assumption that the degree of whiteness of bread bears a relationship to the cleanliness of the article or to its purity in a nutritional sense, and to educate the public with regard to the nutritional benefits that can be achieved by the use of high-extraction flour.

As to the desirability of increasing the supply of wheat offals for feeding livestock, we would suggest that if it is accepted that the nutrients in wheat offals are valuable additions to the human dietary, it is wasteful to give them to livestock and then obtain them at secondhand by the consumption of livestock product It cannot be denied that wheat offals are useful ingredients pig or poultry rations, but it should not be impossible to devis other rations for livestock which are of equivalent nutritive valu

The arguments for and against the continued milling of high extraction flour have to some extent been reconciled by the introduction of new milling methods whereby the particularly desirab portions of the grain can be included in a flour white enough t satisfy all but the most bigoted enthusiasts for white flour.

As we have seen, the germ, including the scutellum, the aleuro layer and the outer endosperm are the parts of the grain riche in nutrients other than Calories, and in order to secure a flou of a low bran content but with the maximum retention of th essential nutrients of the grain, these parts of the grain mu pass into the flour. Having issued instructions to the millers a to how flour should be milled to secure these benefits, the Ministr of Food authorized a reduction in the extraction rate of flour 82½ per cent in October, 1944, and to 80 per cent in Januar 1945, at which level it has since remained.* Flour of 80 per ce extraction has at the most an 'off white' colour, which shou satisfy whatever demand there is for white flour. Its fibre conte of 0·21 per cent is in fact little more than that in white flour 72 per cent extraction. Of more importance, however, is th effect of the reduction in the extraction rate of flour on its nutritive properties. Moran and Drummond (1945) have shown calculations based on the composition of the different parts of th grain that it is theoretically possible to mill a flour of 80 per ce extraction with as high a content of iron and B vitamins as per cent extraction flour. From analytical figures given by Mor and Drummond and also in the Ministry of Food's report, *Natio Flour* (*80 per cent extraction*) *and Bread in Britain* (1945), commercial samples of 80 per cent extraction flour do not, howev achieve those theoretical levels. As is shown in Table 17, loweri of the extraction rate from 85 per cent to 80 per cent has result in a reduction of 29 per cent in the content of iron, 18 per ce in that of vitamin B_1, and 50 per cent in that of riboflavin. If consider the hypothetical case already referred to of a person eati 10 oz. of bread per day, the effect of the change from 85 per cent 80 per cent extraction of flour will have been to reduce the inta of iron by 2 milligrams, of vitamin B_1 by 40 International Units, and of riboflavin by 0·1 milligram.

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* In March, 1946, the extraction rate was raised to 85 per c again, owing to the world food crisis.

Although the reduction in nutrients resulting from the lowering of the extraction rate of flour may be of little importance to those taking a satisfactory mixed diet, this may not be the case for persons belonging to the lower economic classes whose diets are notoriously deficient. Even if the improved milling methods foreshadowed in Moran and Drummond's paper increased the yield of nutrients in 80 per cent extraction flour to that theoretically possible, we still would maintain that 85 per cent extraction flour has definite nutritive advantages, including the laxative properties of its bran content. The truth is that the 80 per cent product constitutes a compromise which satisfies to some extent those who wish to return to white flour and those who, on the other hand, believe in the nutritional virtues of high-extraction flour.

Other Cereals

(*a*) *Rice.* Rice is the cereal crop of swampy regions, and is the staple cereal of the largest proportion of the world's population. It has a lower protein content than wheat, but is otherwise very similar in its dietary properties. It is taken in one of three forms: (1) Undermilled, or brown rice, which contains the thin pigmented layer of adherent pericarp and aleurone layer and also the germ, and is thus equivalent in nutritional value to wholemeal flour; (2) parboiled rice, in which the undermilled rice is boiled and then dried before polishing to remove the pigmented outer coat. In the process of boiling, some of the B vitamins diffuse into the endosperm, so that parboiled rice is intermediate in nutritive value between undermilled rice and the commonest form in which rice is eaten, namely (3) polished rice, in which the pigmented outer layer and the germ are removed by abrasion, eaving only the endosperm. Polished rice therefore has the same nutritional defects as white flour. It forms a larger proportion of the diet among the races who consume it as their staple cereal than does white flour among wheat-eating races, whose diet is more varied. This explains why beri-beri is so common among ice-eating peoples.

(*b*) *Maize.* Maize flourishes in regions where there is a summer rainfall that precludes the cultivation of wheat, but does not produce the swampy conditions in which rice can be grown. It is the staple cereal of large parts of the Western Hemisphere, from which it was introduced into the countries forming th Mediterranean littoral and other areas where climatic conditio are suitable for its cultivation.

Maize has a much lower nicotinic acid content than wholeme flour or undermilled rice, although there is little difference i this respect between highly milled maize and wheat. Whea eating races, however, either consume wholemeal flour or have sufficiently varied diet to prevent the relatively high inciden of endemic pellagra that characterizes maize-eating races. A already mentioned, yellow maize is unique among the cereals containing a significant amount of biologically active carotenoid

Barley and Rye. These are the characteristic cereals of sem arid regions. Their nutritional value resembles that of whea and they are usually eaten in a less highly milled state tha white flour. Their proteins are inferior to the glutenin of whe in dough-forming properties, and bread made from barley or ry is hence harder and inferior in texture to wheaten bread.

Oats. The fact that this can be grown in much more norther latitudes than wheat explains why oats and not wheat is th staple cereal crop in Scotland.

In the milling of oats only the fibrous pericarp and son adherent endosperm are removed, and the germ and other par of the grain rich in minerals and vitamins are retained. Oatme is therefore nearer to wholemeal wheat flour than to 70 per cen extraction white flour in nutritional value. Its protein conte is about the same as that of 85 per cent extraction wheat flou but its energy value is significantly higher, due to the fact tha oatmeal contains much more fat than other cereals. Its vitamin B content is as high as that of wholemeal wheat flour.

Oatmeal has a similar phosphorus content to whole whea but as in the latter, a considerable proportion of the phosphor (about 66 per cent in the case of oatmeal) is present in the for of phytic acid and hence is unavailable to the body. In it preparation for consumption oatmeal is not subjected to bakin which as we have seen leads to the destruction of part of th phytic acid in wholemeal flour in the 'proving' process of brea making. Instead, it is boiled, and this destroys the phytase oatmeal (which in any case is less than that present in whea before the enzyme can inactivate the phytic acid. Hence it migl be expected that oatmeal would have a more deleterious effe on the absorption of minerals than wholemeal bread. Indee oatmeal was one of the foodstuffs used by Sir Edward Mellanb n his demonstration of the rachitogenic property of the phosphorus-rich cereals.

Coarse-ground oatmeal was for centuries the staple cereal of the Scottish peasantry, but their health and physique were certainly not inferior to those of their wheat-eating southern neighbours. Large quantities of milk were consumed along with the oatmeal – a free and generous supply of milk used to be part of the emolument of the Scottish agricultural labourer – and the resultant high calcium intake presumably counteracted any bad effect of oatmeal on the absorption of minerals.

Millet. The different varieties of millet are grown in arid regions where only a drought-resistant cereal can flourish, as in North China and in parts of Russia and India. It is an inferior grain as regards its physical properties, having a very hard seed coat, but in nutritional value it resembles other cereals, except that certain varieties (*e.g.* finger millet) have a very high riboflavin content.

VEGETABLES AND FRUITS

Leafy Vegetables. The value of the green vegetables in nutrition lies in their mineral content, and especially in their high content of certain of the vitamins, which is considerably greater than that of the roots and tubers. The latter are essentially storage organs of starchy food for the young plant of the next generation, whereas the leaves are the actively functioning parts of the plant, whose cells contain all the essential factors for the metabolic processes.

The importance of green vegetables as a source of calcium and iron is perhaps sometimes overstressed. It is true that certain varieties, such as turnip greens, watercress, parsley, spinach and kale are relatively rich in both minerals (although the calcium of spinach is non-available owing to the high oxalic content of that vegetable). But some of the more commonly consumed leafy vegetables such as cabbage, Brussels-sprouts and lettuce do not constitute particularly good sources of the two minerals. If the diet is considered over a period in which several varieties of green vegetables are consumed, the average daily intake from a large helping of green vegetables (6 oz.) would not supply much more than 10 per cent of the optimum intake of calcium and 0 per cent of that of iron.

It is therefore chiefly as a source of vitamins that the green leafy vegetables are of such importance in the diet. They a all rich sources of carotene, which appears to be better utiliz by man than that obtained from the yellow vegetables. T greener and leafier the vegetable, the higher is the carote content; therefore in 'head' vegetables like cabbage and lettu the outer green leaves, often discarded, are rich in carote while the inner white leaves are poor in this substance.

In the fresh, raw state most leafy vegetables are rich sour of vitamin C. As explained in the section dealing with natu sources of this vitamin, a progressive loss of vitamin C occ when vegetables are stored after garnering, especially if they a bruised.

Green leafy vegetables are not on the whole good sources the B group of vitamins. Spinach is, however, exceptional that in the raw state it contains about 0·06 milligram of ri flavin per ounce, *i.e.* 50 per cent more than that in whole mi Since few persons eat spinach regularly or in large amounts high riboflavin content is of little practical significance. Ordin methods of cooking do not affect the vitamin A potency great since carotene is insoluble in water and stable at the temperat reached in cooking. There is likely to be little destruction vitamin B_1 at temperatures used in cooking unless bicarbon of soda has been added to preserve the green colour of the ve tables. The fact that vitamin B_1 is soluble in water leads t loss of up to 50 per cent if the water in which the vegetables h been cooked is discarded. Loss by the same means occurs in case of vitamin C, and the heating involved in cooking also cau destruction of some of this vitamin by oxidation.

The great bulk of the leafy vegetables in proportion to th energy value makes them a useful addition to any diet in wh it is desired to keep the caloric intake low.

Roughage. Satisfactory evacuation of the colon depends many factors, intrinsic and extrinsic. One of the most import is the character of the food residue in the large intestine – so-called roughage – on which depends so largely the stimulat of peristaltic activity. The most suitable residue for this purp is one which is bulky and semi-solid in consistence. It is gener agreed that such roughage is best obtained by the ingestion adequate amounts of food which are rich in cellulose. Cellul forms the cell wall in plants, and because of its resistance to action of digestive enzymes it reaches the colon little chang Owing to its property of absorbing water it becomes softened wollen and hence fulfils the criteria mentioned above. In addition, it undergoes some degree of bacterial fermentation in the olon, with the production of substances having chemically timulating properties. Accordingly, the diet should normally ontain plenty of fruit, green vegetables and the husks of cereals o compensate for the deficiency of roughage in the concentrated nd refined diets so frequently taken to-day.

Legumes. The value of the legumes lies chiefly in the fact that hey are a cheap source of protein. Thus dried uncooked peas, eans and lentils contain about 20 to 25 per cent of protein, hereas green vegetables contain 2 to 3 per cent, potatoes 2 per ent, and cereals about 10 to 15 per cent. The proteins of legumes re of low biological value, however, and must be supplemented y first-class protein.

The legume seeds are for the most part richer in calcium than ther varieties of vegetable. Of more importance, however, is heir high iron content. Weight for weight, dried legumes when ooked contain about half as much iron as meat, but judged by e estimation of ionizable iron by the dipyridyl method, a higher roportion of their iron is available. The legumes, therefore, ffer a cheap and valuable source of iron when the diet is restricted r economic reasons.

The legumes, either fresh or dried, are good sources of vitamin B_1, and, to a lesser extent, of riboflavin and nicotinic acid, but ey are mostly deficient in vitamin A. Fresh legumes are oderately good sources of vitamin C, but dried legumes contain one, unless sprouted on damp cloth before cooking.

The soya bean and the groundnut or peanut (which is actually legume) are peculiar among the legumes in having a relatively gh fat content and therefore a higher energy value than other embers of this group. Their protein content is also somewhat gher, and they are richer sources of the B group of vitamins. aims have been made that the biological value of the proteins oya beans and groundnuts is higher than that of the other gumes, but several workers have found no difference.

he soya bean forms a staple crop in many parts of the Far ast, where it is prepared in different forms (*e.g.* soya bean curd, ya bean milk, etc.) and is eaten as an addition to a rice diet. though it is not a complete supplement to the proteins of rice, e soya bean protein forms the principal source of protein in ch diets. The additional B vitamins provided by soya beans e undoubtedly of major importance in ameliorating some of the deleterious effects of the deficiency of these vitamins diet consisting chiefly of polished rice.

In Western Europe and in America, soya beans are used c as a food for livestock, but in recent years a defatted soya has been made available to the public. It can be mixed wheat flour in baking cakes and buns, so enhancing their nu value.

Tuber and Root Vegetables. These are all fleshy roots or which have been specialized as storage organs for starch. result, they have a considerably higher energy value than vegetables. They are all low in protein, but, in varying among the different members of the group, they are good s of certain of the vitamins.

Tubers. The most important of these are the potato a sweet potato. Both consist largely of starch, but contain a amount of protein of relatively high biological value.

The potato is not rich in minerals, but in the fresh stat tains substantial amounts of vitamin C. As mentioned a when considering the natural sources of vitamin C, th however, a continuous loss of this vitamin on storage, further loss of vitamin C on cooking, although this is les in the case of the green vegetables, unless the potatoes are r or kept hot for a lengthy period after cooking. If properly c potatoes in the amounts usually eaten make an importa tribution to the vitamin C content of the diet. The pota contains a significant amount of vitamin B_1, but is low other B vitamins and deficient in carotene. Sweet pota the yellow variety, on the other hand, are a rich source logically available carotene.

Root Vegetables. These (*i.e.* carrot, turnip, parsnip, bee contain more water and less starch than potatoes, and th have a lower energy value. They provide varying amo vitamin C, swedes and turnips being the richest sources this group of vegetables. They contain only insignificant ties of the B vitamins, and with the exception of the car deficient in carotene. The carrot is peculiar in having high carotene content, but experiments on man suggest th a relatively small proportion of this (varying from 1 to cent according to different workers) is absorbed and uti

Fruits. The nutritive value of the fruits depends on:

1. Their rich vitamin content. This is especially true citrus fruits, currants and berries, the vitamin C content of

r example, being about 15 milligrams of ascorbic acid per ounce. pples and pears have a much lower vitamin C content, ranging om about 1 to 2 milligrams of ascorbic acid per ounce. The loss vitamin C in the cooking of fruits is less than with the vegetables, ving to the acid nature of fruit juice. Also, the juice is con- rned as well as the fruit, whereas much of the vitamin C may lost in the water in which the vegetables have been cooked. rtain fruits, *e.g.* apricots, dried prunes and yellow peaches, are od sources of carotene.

2. Their laxative properties, due to the stimulant effect of the llulose and also of the fruit acids on intestinal peristalsis.

Fruits have only a limited caloric value, containing usually out 10 per cent of available carbohydrate.

Canned Vegetables and Fruits. The increasing use of canned oducts raises the question of their nutritive value in comparison th fresh vegetables and fruits. As regards carbohydrate and tein, there is nothing to choose between the fresh and the nned forms. Canned vegetables are often condemned on account their deficient vitamin content. However, since the introduc- n of modern canning methods, in which the air in the cans is hausted before these are sealed and the contents cooked, and ntamination by copper is prevented, it can be stated that, as urces of vitamin C, canned vegetables and fruits are as good if not better than the fresh products subjected to the usual usehold cooking procedures. In both cases, up to 50 per cent the vitamin C content of the fresh product may be lost.

There is a loss of vitamin C during storage of canned fruits and getables, the extent of which varies among the different products. is deterioration in nutritive value is aggravated by storage at h temperatures. Experiments have shown, however, that with odern canning methods, canned vegetables in which the original tamin C content was high contribute significant amounts of tamin C to the diet even after several months' storage in pical climates.

Canning does not affect the vitamin A potency. There is a rtain amount of destruction of vitamin B$_1$ during the canning ocess, and a progressive loss on storage of the canned product, pecially if storage takes place at high temperatures.

Sir William Savage (1939) has recently published an exhaustive rvey of canned food in relation to health. He comes to the llowing conclusions:

1. Canned foods can be accepted as a safe and convenient

source of food supply and one without any special hazar
nutritional, chemical or bacterial.

2. On the nutritive side, whether judged by the quantitat
or qualitative aspect, canned foods compare favourably w
fresh foods.

A point of importance in practical dietetics is that commercia
canned fruits are preserved in syrup. Therefore the use of can
fruits must be prohibited when either the carbohydrate or
caloric value of the diet is of importance, as in the treatment
diabetes or obesity.

BEVERAGES

Tea and Coffee. The physiological influence of these beverage
due to their caffeine content, which has a stimulating effect
the higher centres of the brain. In moderation, neither is harm
but if taken in excess, toxic effects of caffeine may appear, s
as irritability, insomnia, and tachycardia. Therefore their int
should be restricted in cardiac conditions and in hyperthyroidis
when the heart is already overstimulated. The partaking
excessive quantities of tea, particularly if the leaves have b
stewed for a long time, is a potent cause of dyspepsia (see p. 2

Tea, coffee, cocoa and chocolate all have a high purine conte
and their use must be restricted or forbidden in gout.

Alcohol. Pure alcohol has an energy value of 200 Calories
ounce. The body can completely oxidize only limited quantit
of alcohol, the maximum being 10 c.c. of pure alcohol per ho
the amount contained in about half an ounce of whisky.
advantage is gained by administering alcohol in larger dos
while considerable harm may result from its cumulative acti
In medical practice half to one ounce of whisky or brandy th
or four times daily has been found to be an adequate dose.
small amounts it can be burned in place of quantities of car
hydrate or fat of equal caloric value. Accordingly, any int
of wines or spirits of high alcohol content must be conside
when the caloric value of the diet is of importance, as in obes
and diabetes. The energy intake derived from alcoholic bevera
is not, of course, due entirely to their alcoholic content, si
sweet wines contain unfermented sugar, and beer and other m
liquors contain carbohydrate from the malt.

Beer, cider and stout contain about 5 per cent of alcohol, li
table wines contain 8 to 12 per cent, the fortified wines like p

and sherry contain about 20 per cent, while spirits such as gin, whisky and brandy contain 30 to 45 per cent.

The liqueurs contain 20 to 50 per cent of alcohol, but this is of ittle importance in view of the small amount taken. On the other hand, their sugar content is as high as 30 per cent and a iqueur glassful contains about 5 grams of carbohydrate – a fact to be remembered when a diabetic patient asks advice regarding alcoholic beverages.

A table showing the caloric values of the different alcoholic beverages is given on p. 445.

Alcohol in Medical Dietetics

We are concerned here solely with the problem of alcohol as a therapeutic agent and not with its moral, economic or sociological aspects. The use of alcohol as a medicine has been greatly reduced during the past twenty-five years, and to-day in some hospitals it is scarcely ever prescribed. Accordingly it is advisable to consider whether alcohol should still be classed as a therapeutic agent, and if so what are the indications for its employment. Before doing so, however, it is advisable to dispel once and for ll the popular illusions that alcohol has any specific action in uring or preventing any disease, infective or otherwise, in stimulating the heart, respiration or vital activities, or in improving assimilation and thus increasing bodily strength. The claims of alcohol as a therapeutic agent rest on certain effects which have been ascertained by physiological experiment and clinical observation.

1. Alcohol is a narcotic drug. It is never a stimulant. In its sedative action on the central nervous system lies its chief therapeutic value. Through this effect it blunts the appreciation of pain, relaxes mental tension and reduces the influence of worry nd anxiety. As a result, confidence in ultimate recovery may be induced, sleep promoted, and appetite and digestion improved when these are disturbed by emotional influences.

2. Alcohol is a food. As indicated above, alcohol has a definite but limited food value. Moreover it is absorbed more rapidly than any other food, and its absorption is not dependent on the secretory or motor activity of the alimentary tract.

3. Alcohol dilates the peripheral skin vessels and thus induces mild diaphoresis. The diversion of the blood to the skin may possibly relieve congestion of internal organs to some small degree. This peripheral dilatation induces a subjective sensation

of warmth, but actually aggravates heat loss from the body, ow to the increased amount of blood reaching the surface and th losing heat through radiation, etc. The administration of alco to persons suffering from the effects of exposure to cold and v is therefore contra-indicated until the patient has been broug into warm surroundings and wet clothing has been replaced dry. When the risk of excessive heat loss through peripheral va dilatation has been eliminated by these measures, alcohol may given in the amounts recommended above, to increase t patient's comfort by its sedative effect and by the sensation warmth induced.

4. Alcohol has a mild carminative action, similar but in no w superior to that of the essential oils, *e.g.* peppermint or cloves

On the basis of the above criteria it is possible to indicate t rôle of alcohol in therapeutics.

Acute Febrile Diseases

In its sedative action lies its principal value in such conditio and this is particularly the case if the patient has been acc tomed to its regular consumption. Accordingly for the purp of obtaining mental relaxation and sleep in a worried, anxi patient, alcohol may be of undoubted value in suitably selec cases. When the temperature is high, and appetite and dig tion are seriously impaired, the food value of alcohol, thou limited, is not without importance. It is probable, howev that glucose, which like alcohol is easily absorbed and requi no previous digestion, is preferable to alcohol as a food feverish patients. The carminative and diaphoretic effects alcohol are of value in relieving colic and griping pains a promoting a gentle sweat. It is doubtful, however, if alco should be prescribed for these effects unless its sedative act is also primarily desired.

Chronic Disease and Convalescence from Acute Illness

When worry, anxiety or the tedium of the illness are retardi recovery and impairing appetite and digestion, alcohol may of value for its sedative effects, and this is especially the c in patients habitually accustomed to its consumption. F others, small doses of bromide or barbiturate are preferab In chronic illness it is unwise to prescribe alcohol either for food value, because malnutrition can only be repaired by properly adjusted diet, or for its carminative or diaphore effect. In long-continued ill-health, the dangers of addiction must be remembered.

From what has been said above, alcohol must still be considered a therapeutic agent of limited value in suitably selected cases.

PART III

DIET IN PERIODS OF PHYSIC LOGICAL STRESS

CHAPTER IX

SOME NUTRITIONAL PROBLEMS OF HEALTHY PERSONS

1. IN CHILDHOOD

WHILE the general principles of nutrition mentioned in the p
vious chapters hold good in childhood with even greater force th
in adult life, the diet during childhood requires special conside
tion. No attempt is made here to discuss the diet during infa
and up to the age of two; special feeding methods are necess
during that time, and these are dealt with in many excell
publications. By the age of two, however, the child should
receiving a diet which is in general qualitatively similar to t
of the other members of the family, with certain minor modifi
tions to allow for the following factors:

1. The total food requirement of children is relatively m
greater than that of adults, whether compared on a basis of wei
or of surface area. This large food intake is necessary to sati
the demands made by growth and by the intense physical activ
of childhood.

2. The protein and mineral intake, like that of total Calor
should be relatively much greater than in adults to allow of opti
growth.

3. Vitamin deficiencies produce harmful effects much m
quickly in a growing than in an adult organism. Hence the nec
sity for an adequate intake is of especial importance in childho

Total Food Intake

This must satisfy the demands for growth as well as those m
tioned for adults, namely basal metabolism, activity and
specific dynamic action of the foodstuffs.

The amount of food required in childhood is difficult to de
mine, since the basal metabolism varies with age, the rate incre
ing from birth up to the age of five and then slowly sinking to

dult level. In addition, activity varies greatly in different hildren. It has been estimated that about 36 Calories per pound f body weight daily are required between the ages of two and ixteen years. Any figures given are obviously only very general verages, but Table 6, modified from that given by Holt and ales (1921), may be taken as a rough guide. This table gives the aloric value of the actual food intake of groups of children eceiving an unrestricted diet.

TABLE 6

Caloric Requirements in Childhood and Adolescence

Age Years	Boys		Girls	
	Average weight in pounds	Total daily Calories	Average weight in pounds	Total daily Calories
1 to 3	22 to 32	950 to 1,300	21 to 31	950 to 1,200
3 to 5	32 to 40	1,300 to 1,500	31 to 40	1,200 to 1,350
5 to 7	40 to 48	1,500 to 1,700	40 to 48	1,350 to 1,700
7 to 9	48 to 58	1,700 to 2,100	48 to 58	1,700 to 2,000
9 to 11	58 to 69	2,100 to 2,500	58 to 69	2,000 to 2,500
11 to 13	69 to 84	2,500 to 3,200	69 to 89	2,500 to 2,900
13 to 15	84 to 106	3,200 to 3,800	89 to 106	2,900 to 3,200
15 to 17	106 to 127	3,800 to 3,900	106 to 116	Up to 3,200

One striking point about this table is that the total food require- nt of an adolescent of thirteen to seventeen is in excess of that a moderately active man.

The Composition of the Diet

The Protein Intake must be sufficient to satisfy the demands not ly of tissue wear and tear but also those of the growth of new sue. The daily protein allowance should be about 1·8 grams per und of body weight at the age of two, decreasing to 1·1 grams r pound of body weight for children between the ages of thirteen d fifteen (Orr, J. B., 1939). It should be noted that this means boys between thirteen and fifteen should be receiving from to 126 grams of protein daily – almost 50 per cent more than the lue considered adequate for an adult.

Two-thirds of the total protein should be first-class protein, a at proportion of which should be supplied in the form of milk younger children. If much less than the amounts of milk ommended below are taken, the child's diet will contain too little protein and expert supervision will be needed to bring amount up to the proper level.

The protein intake in childhood is apt to be overlooked in emphasis placed on the mineral and vitamin requirements. Je (1942) has pointed out that the urinary creatinine excretion, wh is directly proportional to the amount of muscle in the body, i useful criterion for estimating the protein content of the bo He finds that in children receiving ample protein the creatin excretion (and consequently the amount of muscle) reaches optimum level, with a narrow range at each age period. Data the creatinine excretion in childhood obtained by himself in United States or from the literature indicate, however, that in great majority of children the creatinine excretion is below optimum level for the corresponding age group, which sugge that the protein intake of children is often too low. The weig of the children were usually within the normal range, from wh it would appear that body weight or size is not necessarily a g criterion for estimating the adequacy of protein intake.

2. *The Carbohydrate Intake.* As in the adult, the carbohydr allowance in the diet should supply 50 per cent of the ener this corresponds to an intake of roughly 5 grams of carbohydr per pound of body weight daily. An adequate intake of car hydrate is essential for its protein-sparing action. It is also cheapest source of energy, a point of great importance in national economy. The tendency, however, is to give yo children an excess of starchy foods and too little animal pro and fat. Other disadvantages of an excess of starch in the are that it may cause abnormal fermentation in the intes and diarrhœa, and that it may lead to excessive deposition body fat.

The carbohydrate allowance should be given chiefly in the f of cereals, potatoes, vegetables and fruit. The sugary foods, s as jam and sweets, should be given in the form of simple dess at the end of a meal; eaten between meals they destroy the appe for more essential foods and may cause alimentary upset. excessive amounts, sugary foods are believed to be a potent fa in the production of dental caries.

3. *The Fat Intake.* This should vary from 1·5 grams per po of body weight in young children to 1·8 grams per pound of b weight in active children about the age of fifteen. A liberal intake must be provided to secure an adequate supply of the soluble vitamin A, which is essential to growth. Fat should

given in the form of butter, vitaminized margarine, cream and egg yolk, rather than in fried foods.

4. *Minerals and Vitamins.* If milk, fruit, wholemeal cereals and vegetables are taken liberally, the only essential nutrient that will not be supplied in adequate amounts will be vitamin D.

The mineral requiring chief attention is calcium, since the other essential minerals are likely to be present in sufficient amounts, especially if the diet includes bread made from high-extraction flour (see p. 186). The efficiency with which children utilize ingested calcium varies widely unless a supplement of vitamin D is given, when the range of retention for each age group becomes small. Calcium is not very readily absorbed, and even when vitamin D is given, the amount ingested greatly exceeds the amount retained. The daily retention requirement is about 300 milligrams in infancy, decreasing to below 200 milligrams in the pre-school period and then increasing to about 450 milligrams in early adolescence, but to secure such retentions, the child's diet must contain about 1·0 gram of calcium and that of the adolescent up to 1·5 grams.

Milk and milk products are the richest food sources of calcium. The usual child's diet is not likely to contain more than 0·2 o 0·3 gram of calcium apart from milk and milk products, from which the remainder of the requirement of 1·0 to 1·5 grams must e met. Up to the age of five years, about a pint of milk daily will aise the calcium content of the diet to about the theoretical quirement; from five to twelve years of age, the child should take ½ pints daily, and from twelve to sixteen (or twenty in the case f youths), the daily intake must be 2 pints if the theoretical alcium requirement is to be supplied by the diet.

Milk should not be considered solely as a source of calcium. It upplies other valuable nutrients such as vitamin A and riboflavin, nd, as already mentioned, the diet is not likely to be adequate protein unless milk is ingested in the amounts mentioned bove.

Calcium deficiency, at least in moderate degree, is believed to be revalent in childhood, and is almost certainly worse during dolescence, when the requirement is at its highest, and the intake all too frequently reduced by a decrease in milk consumption. is during the adolescent period particularly that dental caries ends to become serious, and for this development calcium defici- cy is believed to be one of the responsible factors.

A diet which includes the recommended milk ration, and generous amounts of vegetables, fruit and wholemeal cereals, v have an adequate content of vitamins other than vitamin D. T normal diet contains only small amounts of vitamin D, and compensate for this there is seldom quite enough sunshine summer, let alone in winter, in such climatic conditions as th prevalent in Britain. The administration of a vitamin D supp ment has become a routine in infant feeding, but it is not genera realized that such a supplement is probably required througho the growth period. Without special methods of investigation, i impossible to detect those children whose utilization of calci and phosphorus, unaided by a vitamin D supplement, is poor, a it is therefore wisest to assume that all children require ex vitamin D. A supplement of about 400 to 800 International U daily will produce satisfactory calcium and phosphorus retenti provided that the intakes are adequate, and this amount sho be given up to the age of twelve years, at any rate during winter months. It is usually assumed that after this age child will obtain all the vitamin D they require from the action of s light. As pointed out in Chapter IV, however, the occurrence cases of late rickets in adolescence indicates that this source vitamin D may not be adequate in every case, and ideall vitamin D supplement should be continued throughout adolesce to ensure an adequate calcium retention during this period particularly rapid growth.

Until recently it was widely held that the essential factors c cerned with the satisfactory development of bone and teeth the prevention of dental caries were calcium, phosphorus vitamins A and D. It is now believed that at least some of features claimed to be characteristic of rickets are in all probabi due to a deficiency of vitamin C. It is therefore extremely portant during infancy and childhood to make sure that an an supply of vitamin C is taken (see p. 80).

The Arrangement of the Child's Diet

As regards meal times, it is probably best to give the child o three meals a day up to the age of about ten. The child approach these meals with a healthier appetite than if he is down to small and frequent feeds, and there will be less chanc over-feeding. Provided a good breakfast is eaten there is no n for a mid-morning ration of milk and biscuits. This, however, r be justified in schools in which the pupils are drawn from p homes. After the age of ten, supper should be introduced as we tea' as an additional meal. At both supper and 'tea' milk should e the principal beverage supplied.

The general management of the child as regards adequate sleep, ut-door exercise, etc., is important to ensure a healthy appetite. While irrational dislikes to certain essential foods may have to be onquered, undue urging or coaxing of the child at mealtimes is o be avoided. Food such as milk, which is refused at one meal, nay be taken readily later in the day under the stimulus of ncreased appetite.

The first dentition is complete before the age of three, so that rom this time onwards the child is well able to chew and masticate is food, and there is no need to make the diet any softer in composition than that of older children or adults. In fact this is efinitely undesirable, as chewing is necessary for the normal evelopment of the jaws.

Milk is the most important food for the growing child, and his iet must be built up around the milk allowance. It is unnecessary give the child his whole daily ration of milk as a drink – me of it should be given in the form of cocoa, custards, junket, heese, etc.

Eggs form a valuable food for the growing child, and at least ne should be eaten daily.

Meat. In young children, the amount given should be small, to nsure that the appetite is not satisfied without the inclusion of e daily milk allowance. The meat should be tender and cut up ely.

Older children can be given more liberal helpings of meat, but is should be simply cooked, and no highly seasoned meat dishes ould be offered.

Cereals. These are excellent foods for children, especially if they e served with milk. Wholemeal cereals such as brown bread or tmeal are preferable to more refined products, owing to their her mineral and vitamin content.

Green Vegetables and Fruit. At least one meal a day should ntain green vegetables. Tomatoes, too, are valuable on account their high content of carotene and vitamin C. Fruit likewise ould be served at least once daily, especially the citrus variety. re should be taken that raw fruit is fully ripe.

Sugary foods, such as sweets and preserved fruit, are harmless given only in moderation, and at the proper time, which is at e end of a meal.

An example of a diet suitable in amount for a child of five is

Diet No. 38, p. 500. For younger or older children, the amou should be varied according to the general rules mentioned in chapter.

2. IN PREGNANCY AND LACTATION

The diet during pregnancy and lactation must provide the nor physiological food requirements of the mother together with s additions as are needed for the growth of the uterus and for nutrition of the offspring during intra-uterine development an early infancy. Both purposes correspond with perfectly nor physiological functions and any faddism in regard to diet is q unnecessary. It is not enough, however, to dismiss the questio diet for the expectant mother by simply saying that her n are those of any healthy woman, for although she should eat same foods as any other adequately fed woman her intake n be adjusted to enable the infant to develop without any d on the maternal resources.

The fœtus may be regarded as a parasite, since it takes w nutriment it needs, even at the mother's expense, if the di inadequate for both. This is well illustrated by the fact that birth weight of infants is little influenced by any maternal die restriction short of actual starvation.

While it is true that the fœtus obtains the material it requ at the maternal expense, it cannot obtain something the mo does not have, and specific maternal deficiencies, when sev may be exhibited by the newborn infant or may develop sho after birth. For example, fœtal rickets has been describe occurring in countries where the calcium content of the di grossly deficient and the exposure of women to sunlight is stricted, while infantile beri-beri has been reported among bre fed infants whose mothers' diet was grossly deficient in ane

Although such gross nutritional deficiencies are extremely in European countries under normal conditions, minor nutriti deficiencies undoubtedly impair to varying degrees the health efficiency of a certain proportion of the people. Such n deficiencies, which may never incapacitate the normal indivi may be aggravated by the increased nutritional needs of p nancy to an extent that makes them clinically recognizable. example of this is the frequent occurrence during pregnan minor signs of vitamin B_1 deficiency, sometimes amounting t actual polyneuritis.

While, therefore, it is true that pregnancy is a normal physiogical process that does not call for any elaborate dietary precriptions, it definitely imposes a nutritional strain, and the dietary eeds of the expectant and nursing mother are greater than those f the non-pregnant woman. This fact cannot be overstressed, for umerous surveys have indicated that the nutritional state of the other is a very important etiological factor in both fœtal and aternal disasters. Thus, Balfour (1938) has reported a largeale experiment in England, wherein needy mothers, receiving an adequate diet, were provided with supplementary milk and egg roducts fortified with vitamin A and D and a vitamin B conntrate. Among several thousand women receiving these suppleents, the maternal death rate was 1·66 per thousand total births, s against a rate of 6·15 per thousand among women in the same stricts during the same period who did not receive the suppleentary foods. The number of still-births and neonatal deaths as 43 per cent less in the treated than in the control group.

A similar but smaller experiment has been reported from Canada y Ebbs, Tisdall and Scott (1941). A group of 210 pregnant women own to be taking very poor diets was divided into two groups, e of 120 acting as controls to the other group of 90, which was pplied with dairy products, oranges, tomatoes, wheat germ, and tamin D capsules, and was instructed in the proper type of diet r pregnancy. Both groups were followed throughout the second lf of pregnancy and during the puerperium. The incidence of scarriages, premature births, still-births, and infections was less, d the mental and physical condition of the mothers was better, those who received the extra food; the general condition of the bies born of these mothers was likewise much better and the idence of illness among them much less than in the control oup.

In the light of these studies it is clearly the doctor's duty to plain the dietary problems of pregnancy to the expectant her and to instruct her in the types and amounts of food she ould eat in order to obtain the essential nutrients in the necessary antities.

The estimated dietary requirements during pregnancy are given Table 1 on p. 2. It has been pointed out elsewhere that the owances recommended for several of the nutrients in this table ust be regarded as provisional estimates only, as the experiental evidence required to fix standards is as yet incomete. This qualification holds with especial force for the pregnant

woman. The requirements of some of the nutrients such
minerals and protein have been assessed from metabolic bala
experiments, but this is not possible with other of the nutrien
Consequently it is unwise to define the dietary requirements of t
expectant mother within too narrow limits, and the figures quot
in Table 1 must therefore be regarded only as guides and not ha
and-fast standards.

With these reservations in mind, it is necessary to consider h
the essential dietetic requirements are modified by pregnancy a
lactation.

Calories

During the first half of pregnancy the basal metabolic rate rema
unchanged, and there is accordingly no increase in the fuel requ
ments. But even these normal requirements may be difficult
maintain in view of the nausea and lack of appetite that
infrequently mark the first three months of pregnancy.

After the fourth month, however, the basal metabolic r
increases until at term it is almost 25 per cent greater than in
non-pregnant state. The resulting increase in the caloric requ
ments is to some extent compensated for by the necessary rest
tion of muscular activity during this period, but it is desirable t
the intake should be increased to about 2,500 to 3,000 Calo
to meet the demands of the increased metabolism. This incre
is usually ensured by the increased appetite that is a nor
feature of the later months of pregnancy.

Protein

The developing body tissues of the offspring and the increas
size of the uterus both demand protein material, so that the prot
requirements are significantly enhanced during pregnancy
lactation. Varying figures for the optimal protein intake of
expectant mother have been given, ranging from 85 to 120 gr
per day; but whatever the actual figure, the importance of prov
ing an ample supply needs no stressing. At least half of the t
protein intake should be of high biological value and must there
be derived from animal sources. This will be secured by
moderately large serving of meat daily in addition to the amour
milk recommended below.

There is no justification for the belief that a restricted pro
intake during pregnancy is necessary to 'spare the kidneys'
that such restriction will lessen the susceptibility to renal c

lications. Clinical experience does, however, lean to the view
hat toxæmia of pregnancy, once developed, should be treated by
rotein restriction. An increase in protein in the diet tends to
ncrease the yield of breast milk – an ample intake of protein in
he last months of pregnancy thus improving the chances of
uccessful lactation.

Calcium, Phosphorus, and Vitamin D. The metabolism of these
ree substances is so intimately linked that they are best con-
dered together.

Calcium is one of the most important elements of the diet during
regnancy and lactation. Over 60 per cent of the calcium and
hosphorus content of the full-term infant is obtained in the last
o months of pregnancy. The urgency of this demand for calcium
shown by the fact that in the last months of pregnancy there
nds to be a fall in the serum calcium with a return to normal
ter delivery, this fall being more pronounced when pregnancies
ceed each other at only short intervals.

The requirements are met by a daily intake of 1·5 to 2·0 grams
calcium. To this end, as well as to ensure a desirable intake of
her essential nutrients, it is recommended that the diet of the
egnant woman should include a quart of milk per day, part of
hich may be taken in the form of cheese, one ounce of which
ay be regarded as equivalent to seven ounces of milk. A quart
milk will supply almost 1·4 grams of calcium, and the remainder
the requirement will be obtained from the rest of the diet.
here is no need to prescribe a special supplement of calcium if
e expectant mother can be induced to take a quart of milk daily.
is is not always an easy matter, as women in Great Britain at
y rate do not readily take milk in such amounts. Tactful per-
asion by the doctor is thus often called for. It is worth reminding
patient that liquid milk can be taken in the form of milk
kes, milk puddings, milky cocoa or chocolate, etc.

Phosphorus need not be considered in detail, since a diet
equate in calcium automatically supplies the requirements for
s element too.

The amount of vitamin D necessary for adult mineral
tabolism is, as we have seen, unknown. It has been shown in
mal experiments that, when the maternal diet is adequate,
amin D causes an increased retention of calcium and phosphorus
the offspring.

Jeans and Stearn (1938) have stated that pregnant women can
ain calcium in ample amounts on intakes ranging from 1·6 to

2·5 grams, even without added vitamin D, but that in rapi succeeding pregnancies and periods of lactation there may be p retention of calcium and phosphorus regardless of intake, unl a vitamin D supplement is given. It is therefore advisal although conclusive experimental evidence is perhaps lacking, t the expectant mother should receive such a supplement through pregnancy, and the amount empirically advised is 400 to International Units per day, given in the form of any of preparations mentioned in Chapter IV.

The amount of calcium and vitamin D in the maternal affects the density of the infant's bones and the structure of teeth. There is considerable evidence that rickets develops ear and more frequently in the offspring of mothers whose diets dur pregnancy contained inadequate amounts of calcium and vita D, and also that structural defects in the teeth of the first dentit and dental caries are more frequent in such offspring. The ca factor in dental caries is still uncertain, but studies of the incide and control of this condition in pregnant women suggest t dental caries is increased during pregnancy and that diet is important etiological factor.

Iron. A daily intake of 15 milligrams of iron will produc positive balance in most pregnant women, and it is possibl supply these needs by diet alone if the woman has not ent pregnancy with a pre-existing anæmia. Fœtal demands are significant until the last three months, when they form a consi able proportion of the total daily intake, amounting to alm 7 milligrams per day in the last month of gestation.

Many women, however, especially in the lower economic gro begin pregnancy with a pre-existing iron deficiency due to chr blood loss from menstruation and a low dietary intake, the increased demands of pregnancy precipitate an anæmi aggravate a pre-existing one. The most important causes of i deficiency anæmia in pregnancy are as follows:

(1) transfer of maternal reserves to the fœtus;
(2) poor diet;
(3) previous loss of iron reserves through rapidly repe pregnancies, twin pregnancies, or excessive menstrual b loss;
(4) gastric hypoacidity or anacidity during pregnancy, w may be responsible for failure of iron absorption even in presence of adequate supplies.

It must be emphasized that, once the anæmia is established, ifficult to raise the hæmoglobin level by diet alone and iron ierapy must supplement the provision of a nourishing, iron-rich iet. It is a wise precaution to anticipate the aggravation of a re-existing iron deficiency by a determination of the hæmoglobin vel early in every case of pregnancy, and to prescribe an iron upplement wherever this is indicated by the results of the test.

itamins

he requirements of most of the vitamins are thought to be in- reased during pregnancy, and as the body has little power of toring vitamins apart from vitamin A, this increased demand ust be met from the diet.

(1) *Vitamin A.* Recent studies by Lund (1945) have shown that pregnancy, whether the initial plasma vitamin A level is normal r sub-normal, the level tends to fall during the course of the regnancy, although this fall occurs later when the diet is good ian when the diet is poor. Within twenty-four hours of delivery, ere is a rapid elevation of the plasma vitamin A to levels well bove those present at the beginning of pregnancy. The fall in the lasma vitamin A level during pregnancy cannot be due to fœtal emands alone, for it occurs even when large doses of vitamin A re given in addition to a generous diet. The significance of these hanges is not known, but they suggest a storage of vitamin A uring pregnancy and a post-partum release for lactation. In this nnection, it is of interest to note that the infant is born with ery low vitamin A stores, but that the colostrum and early uman milk are about 5 to 10 times as rich in vitamin A as the ter milk.

The rôle of vitamin A in the development of the fœtus is not nown, but its importance is suggested by the fact that, in experi- ental animals, a deficiency of vitamin A in the maternal diet uses defective development of the teeth of the offspring, and an creased rate of premature births and still-births.

Until the optimum extent of the maternal storage of vitamn A escribed above has been elucidated, it is impossible to state finitely what the needs of pregnancy are. Various estimates ave placed the requirements at from 4,000 to 5,500 International nits of preformed vitamin A per day. Although these levels can secured by diet alone – *i.e.* if generous helpings of carotene-rich getables are taken in addition to the milk recommended above – is generally considered advisable to prescribe a vitamin A pplement of about 5,000 International Units per day as well.

This can of course be provided by the same preparation a prescribed for vitamin D, by the use of cod-liver oil or of one of vitamin A and D concentrates mentioned on page 99. Dur lactation, the concentrate prescribed should contain prefor vitamin A and not carotene, as the former appears to influence vitamin A content of the milk more than the latter, and it is better absorbed and utilized by the infant.

(2) *Vitamin B_1.* The vitamin B_1 requirement depends on level of metabolism and the caloric intake. Since these increased in pregnancy and since the infant's vitamin B_1 quirements have to be met from the mother's milk, it foll that the vitamin B_1 requirements must be higher in pregnancy lactation than in the non-pregnant state. Various estimates of requirements during pregnancy have been made, ranging fr about 600 to 1,000 International Units per day, *i.e.* from two three times the minimum requirement in the non-pregnant st

The higher values recommended are practically unattain from dietary sources alone even if wholemeal bread is eaten, it may therefore be advisable to prescribe a supplement of of the vitamin B_1 preparations mentioned in Chapter IV.

Mention has already been made of the development of sign vitamin B_1 deficiency during pregnancy, due to the enhan vitamin B_1 requirements which produce a conditioned deficie Several workers have commented on the correlation betw symptoms such as fatigue, cramps, nausea and vomiting in p nant women and the consumption of diets with an inadequ vitamin B_1 content, and on the improvement in the physical mental condition of pregnant women on poor diets when t vitamin B_1 intake was doubled or trebled.

Some writers believe that vitamin B_1 deficiency is an etiolog factor in such complications of pregnancy as hyperemesis gr darum and other toxæmias of pregnancy. Claims have been m for their successful treatment with doses of vitamin B_1 in order of 10 to 20 milligrams per day, injected parenterally, these have been disputed.

Whether or not there is a causal relationship between vitamin deficiency and hyperemesis gravidarum, prolonged and sev vomiting of pregnancy leads to a deficient intake of all nutrie including fluid, salts, and vitamins. If parenteral administrat of glucose is employed in the treatment of hyperemesis gravidar it should be remembered that the needs for vitamin B_1 are ther increased, since this vitamin is required for the metabolism lucose. It is therefore rational treatment to give the water-oluble vitamins parenterally as part of the treatment of hypereesis gravidarum.

(3) *Riboflavin.* This vitamin is associated with cellular oxidation rocesses, and the increased metabolism in pregnancy probably icreases the riboflavin requirement. The estimated requirement f 2·5 to 3·0 milligrams can readily be obtained from dietary ources alone, the recommended daily quart of milk alone upplying about 1·6 milligrams.

(4) *Nicotinic Acid and the remaining members of the vitamin B omplex.* These vitamins have not been demonstrated as having ny particular significance during pregnancy, but there is doubtless n increased requirement for those concerned with cellular oxidaon processes. This can be met from the diet alone provided that contains adequate amounts of meat, milk, eggs, and whole-grain roducts.

(5) *Vitamin C.* The plasma vitamin C level tends to be lower an normal in pregnant women and falls somewhat towards the d of pregnancy. Pregnant women have been shown to need rger doses of vitamin C than normal adults to maintain excretion vels considered by the proponents of saturation tests to indicate adequate intake. Regardless of the interpretation of these sts, which has already been discussed on pages 76 to 77, the bove evidence points to an increased requirement of vitamin C pregnancy and lactation. The extent of this increase has been ssessed on the results of saturation tests, and on this basis the aily requirement of vitamin C has been placed at 100 to 150 illigrams. Evidence is no more convincing that saturation reprents the optimum nutritional state in pregnancy than in the on-pregnant woman, and these allowances therefore probably err the side of generosity. A vitamin C intake of 100 milligrams per y can readily be obtained from the diet if this contains liberal pings of fresh vegetables and citrus fruits or their juices; no m can come from consumption of vitamin C in excess of the tual requirements, so that it is advisable to aim at this figure as intake during pregnancy. It is of interest to note that the fœtal ood almost always contains more vitamin C than the maternal ood, irrespective of the level of the vitamin in the latter.

During lactation, the nursing mother of course needs enough tamin C to satisfy the requirements of both herself and her child. he vitamin C content of human milk cannot be raised above bout 8 milligrams per 100 cubic centimetres, however much vitamin C is given to the mother, and if this represents optimum level, then the nursing mother requires upwards o milligrams of vitamin C daily to provide for the vitamin secr in the milk, in addition to her own requirements. Therefore diet during lactation should contain at least 100 milligram vitamin C per day. Even on low vitamin C diets, nursing mot will secrete enough of the vitamin in their milk to maintain infant's plasma vitamin C level at a higher level than their own, scurvy among breast-fed infants is consequently extremely ra

(6) *Vitamin E.* There is no more than presumptive evide that this vitamin is needed for normal pregnancy. The requ ments if any are unknown, and its widespread distributio foodstuffs makes any serious deficiency unlikely.

(7) *Vitamin K.* There is no indication that the normal sou of supply of vitamin K are likely to prove inadequate du pregnancy, but the administration of 1 to 2 milligrams just be the onset of labour or, failing that, early in labour, undoubte reduces the incidence of hæmorrhagic disease in the newborn, building up the fœtal vitamin K content in readiness for critical early neonatal period (see page 123).

SUMMARY

Pregnancy and lactation are normal physiological states which not require elaborate dietetic regimes. The diet should, howe supply more of the essential nutrients – especially of prot calcium, and vitamins A, B_1, C and D – than are needed by non-pregnant woman.

The diet will provide the recommended amounts of the differ nutrients, except for vitamins A and D and perhaps vitamin if it includes the following foods daily:

Milk	1½ pints
Cheese	1 oz.
Butter (or vitaminized margarine)	2 oz.
Egg	1
Meat	1 liberal helping (live once a week)
Potato	1 average helping
Green vegetables	1 liberal helping
Other vegetables	1 average helping
Orange or grapefruit or their juice	3 to 4 oz.
(or tomatoes or tomato juice	7 oz.)
Wholemeal bread or bread made from high-extraction flour	4 slices
Wholemeal cereal	1 average helping

Extra Calories necessary can then be obtained from the other foods as required.

A daily supplement of 1 to 2 teaspoonfuls of cod-liver oil, or of a vitamin A and D concentrate supplying about 5,000 International Units of Vitamin A and 400 to 800 International Units of vitamin D should be prescribed. Where there is any reason to suspect that he pregnant or lactating woman is not receiving at least 600 nternational Units of vitamin B_1 daily, a supplement of one or other of the vitamin B_1 preparations mentioned in Chapter IV hould also be given. If hæmatological examination shows that ron-deficiency anæmia is present during pregnancy, a suitable preparation of iron should be prescribed.

Constipation in Pregnancy. One of the troublesome complicaions of pregnancy in its later months is constipation. This can be reated by the methods suggested on pp. 288 to 290.

3. IN ATHLETIC TRAINING

n no sphere of dietetics has faddism and ignorance been more onspicuous than in the field of athletics. As aptly put by Adolphe Abrahams (1934), "Diet has been credited with ttributes and possibilities always exaggerated and generally maginary." The dietetic requirements for the athlete in raining are based on the same fundamental principles which overn the nutrition of human beings in general as outlined in he section on Physiology (p. 17). The energy requirements of he athlete are proportional to the severity of the exercise underaken in training and are, in general, considerably higher than he figure of 2,500 to 3,000 Calories accepted as the average for ormal persons doing ordinary work. Dietetic surveys have hown that individuals, such as lumbermen and navvies, who are ngaged in severe physical exercise take from 4,000 to 5,000 alories a day. A similar intake is required for athletes in hard raining. To attain this figure the carbohydrate, fat and protein f the diet must all be increased, but the ratio between them hould remain unaltered. Quantities of protein up to 200 grams aily are regularly eaten both by lumbermen and by athletes uring training. Not only is there no evidence that such high rotein intakes are harmful for those actively engaged in strenuous xercise, but informed opinion is in favour of the view that they re actually necessary under these conditions for the maintenance f a high standard of physical fitness. It is obvious therefore hat, provided the requirements for protein, carbohydrate, fat, vitamins and minerals are properly satisfied, the diet for athlete in training differs only from the ordinary well-bala diet in that it must provide an extra source of energy. S energy can be obtained from protein, fat or carbohydrate, apparent that there can be no logical reason for claiming any single article of food or any special diet has some partic merit for the training of athletes. The quantity of food e daily should not be calculated by intricate scientific form since it is best assessed by the individual's natural appetite. idea that muscular strength or stamina can be obtained 'stuffing' the individual in training with huge quantities of f cannot be defended on any dietetic grounds. Equally unso is the view advocated by some trainers that fluids should severely restricted during training. In fact the reverse is the since hard exercise causes serious loss of fluid from the b which accordingly should be well stocked before exercise is tal

It is also necessary to consider the best time before a con for an athlete to take food and the most suitable compositio the meal. Since sugar is the most easily absorbed and most rea utilizable foodstuff, it has been particularly recommended recent times as a source of energy to be taken immediately be a race or during a test of the long-distance Marathon t Abrahams says that it is doubtful if the claims as to the benefi effects of sugar can be substantiated, but concedes that practice is at least harmless. We feel that the position in reg to food taken before an athletic contest has been clearly sta by Abrahams as follows: 'It may be stated dogmatically that final meal before a race is dictated by the experience of the k and quantity of food that have been found to permit of extr exertion with the maximum of comfort. Individual requireme vary enormously and it is folly to dictate any more general po than that whatever is preferred should be taken at least t hours before the race is due.'

The relation of diet to training may be summarized by say that a plentiful, well-balanced diet should be taken, due c sideration being given to the individual's inclination and natu appetite. Indigestible articles of food (see p. 265) such as n bread, pastries, suet puddings, and coarse green vegetables, a excessive quantities of fluids at meals should be prohibit otherwise the resulting flatulence may cause respiratory a cardiac embarrassment.

CHAPTER X

NUTRITION AND DIET IN WAR-TIME

HE completion of six years of war makes the time opportune
r a review of the British Government's war-time nutritional
licy, the scientific basis on which it was developed, and its effects
the nation's health. Ample evidence has been collected by Orr
d others to show that a considerable section of the population
as living, before the Second World War, on a diet which was
adequate to meet the optimal demands for growth and the
aintenance of health (see Chapter I). It is also clear that the
etary deficiency was mainly qualitative rather than quantitative.
other words, the caloric value of the diet was seldom at fault;
e deficiency was essentially of vitamins, minerals, and, to a less
tent, of first-class proteins.

In arriving at any conclusion about the effect of war on the diet
the nation as a whole, two questions will have to be answered:

(1) Have the restrictions imposed by war produced an undesir-
le reduction in the quality or quantity of the diet consumed by
ople whose intake of food was formerly unrestricted by
onomic circumstances?

(2) Have the war-time food measures produced an improvement
a further deterioration in the diet of that section of the popula-
n which before the war was unable to buy enough of the right
nd of food to supply its nutritional requirements?

The second question is the more important, for the people in this
tion of the population had no nutritional 'slack' to take up, and
y further deterioration in their diet would necessarily have led
such a serious increase in ill health that the morale and efficiency
the home front would have been undermined.

Before attempting to answer these questions, it would appear
irable, first, to consider the effect on the nation's food supplies
ich resulted from Germany's occupation of Europe, and the
uction in the available shipping by hostile attack, and by its
cation for carrying troops and munitions; and secondly, to
cuss the Government's war-time policy to offset these difficulties.

anges in Food Situation produced by the War

ce two-thirds of Britain's food was imported it was obvious that
erious reduction in our food supply would result as soon as the

stocks of imported foods were used up, unless drastic mea were instituted both to augment the supply of ships and to inc the production of certain foods, the balance being imported ac ing to a plan designed on a scientific basis. The country was f not only with a deficiency of such basic products as meat, fats, cereals, but also of important protective foods such as the products, fruit, and vegetables.

Food Policy to Meet these Changes

The Government of Great Britain in 1939 had the advantage its predecessor of 1914–18 of having available a large amou information about the production, distribution and consum of food both in this country and in Germany and Austria du the First World War. This subject has been admirably dealt by Professor Marrack in his *Food and Planning* (1942). It is apparent that the state of nutritional deficiency which result a breakdown of the home front in Germany and Austria attributable as much (or more) to the reduction in the ca value of the diet as to its qualitative deficiencies. By 191 official ration provided about 1,250 Calories daily in Germany a considerably lower figure in Austria. The purchase of rationed food only raised the caloric value by from 300 to Calories daily. It is not surprising, therefore, that the standard of production by the workers, and the morale of the home f steadily declined.

With this object lesson in mind the British Government de that it was essential in the first place to secure the caloric val the nation's diet, and accordingly took steps to increase gr the home production of food and to import the remaining req ments according to a plan designed on a scientific basis. It necessary to decide whether the land should be used for produc of crops which can be used directly for human food, or for production of foodstuffs for animals. The answer to this que is clear, since it has been proved beyond doubt that animals a poor return for the food with which they are fed. Marrack s that a steer requires 16,200 Calories of feeding stuffs to pro 1,000 Calories in human food. Sheep and pigs require 9,000 5,500 Calories respectively. Put in another way, an acre of pota for human food produces four million Calories, an acre of w two million Calories, while an acre of grass for feeding beef c produces only 200,000 Calories. An agricultural policy w concentrated primarily on the production of cereals and pota as therefore adopted as the correct one. By 1943 the production wheat, barley and oats in tons had been increased by 80 per cent nd of potatoes by 95 per cent.

The great increase of land under the plough resulting from this licy was inevitably accompanied by a corresponding decrease of nd under pasture. Since milk cannot be satisfactorily replaced any food and cannot be imported from a distance in a fresh ate, the maintenance of, or even an increase in, milk production as accepted as another essential feature of the war-time agricul- ral policy. This could only be accomplished by carefully control- g the ploughing-up policy on dairy farms and by giving dairy ttle the highest priority in the issue of concentrated feeding uffs. No issue of feeding stuffs was provided for beef cattle which ould be competing with the milk cows for the reduced amount of sture available. Very restricted rations were granted for pigs and ultry in order to conserve as much of imported foodstuffs as ssible for dairy cattle, and in consequence a material reduction their numbers resulted. That this policy has been satisfactorily complished is clearly indicated by the fact that the production milk has been maintained at the pre-war level, despite the great uction of grass land. The consumption of liquid milk was tually 34 per cent higher in 1944 than in 1938, because the version of liquid milk to manufacturing purposes had been ohibited.

The almost complete cessation of imports of fruits and vegetables s obviously a factor of great importance which had to be taken o consideration in designing the food policy, since pre-war tary surveys had clearly indicated that a considerable section the population was receiving insufficient quantities of vitamins and C. Accordingly, the third essential feature of the food policy s an increased home production of green and yellow vegetables. 1943 an increase of 33 per cent over the pre-war consumption d been secured by a combination of methods, including stabiliza- n of prices and subsidies for the professional grower and propa- nda directed towards the private individual with a garden or tment.

port Programme

matter how greatly home production is increased in Great tain, it can never meet the nation's requirements, and accord- ly the importation of food is as vital in war-time as in peace- ne. The shipping shortage made it essential, however, that imports should be reduced, and the most careful consideration to be given to the question which foods should have priority assessing this aspect the foodstuffs had to be considered not from the nutritional point of view but also with respect to sto transportability, and the length of journey they would ha travel. The first priority on shipping had obviously to be res for wheat, which is easily transported and stored and which stitutes the most suitable food for maintaining the caloric nee the nation. The Government's decision to keep flour, bread potatoes unrationed as long as humanly possible was undoub sound both on administrative grounds and because it ensured no one needed to go hungry for want of energy-giving foods. second priority was reserved for fats, which have more than the energy value of carbohydrates, are valuable sources of soluble vitamins, and take up relatively small amounts of space. The third priority for importation was cheese, which particularly condensed form of food, valuable for its fat, cal and protein content. The latter aspect was of special impor because of the decision greatly to reduce the importation of Meat is a foodstuff which is bulky and which requires speci frigeration facilities. The importation of fruit had to be aband almost completely because of its bulk and poor keeping qualit

With the object of increasing the carrying capacity of the available for the transport of food, the bulk of the foodstuffs wherever possible, reduced by the processes of compression dehydration (*e.g.* milk, eggs, fruit juice, and, more recently, and by deboning the carcasses of animals. By increasing extraction rate of flour from 70 per cent ('white flour') to 8 cent (National wheatmeal flour) a greater yield of flour obtained from a given weight of grain. The introduction of t per cent extraction flour in 1942 not only saved several hu thousand tons of shipping space annually but secured the vit B requirements of the nation and greatly augmented its su of iron and valuable vegetable protein. The results of the Go ment's policy on the Nation's diet after nearly five years o can be clearly seen in Tables 7 and 8 (see p. 227), which are structed from data published in *Food Consumption Levels* (1

A study of these two tables shows that the caloric value o diet was maintained by an increased intake of cereals and pot which offset the reduced consumption of meat and fats. The protein content of the diet was increased by 7 per cent large a result of the increased intake of vegetable protein in the nat af. The carbohydrate content of the diet was approximately ationary, and only in the intake of fat was there any material crease. An increase in the intake of calcium to a highly satisfac- ry figure of 1 gram daily resulted from the increased consumption milk and cheese and the fortification of the National wheatmeal our with calcium. The increase in the iron content of the diet, d the bringing of the vitamin B_1 and B_2 complexes up to a safety vel, can be attributed mainly to the use of 85 per cent extraction our. With regard to the figures for vitamin C, these must be cepted with caution. A high proportion of vitamin C in pre-war ys was derived from imported fruit eaten in the fresh state. In e war-time diet vitamin C was, and continues to be, derived rgely from vegetables and potatoes, and has been subject to great terioration in the course of transport, storage and preparation the kitchen and during cooking. It is also probable that the take of vitamin A by a section of the population has fallen below e optimal level, for since the war the source has been essentially rotene from green and yellow vegetables, and it is now believed at, unit for unit, carotene is much less valuable than vitamin A.

In conclusion, it may be said that, although the war-time diet s been monotonous, it has been adequate with respect to Calories d protein and superior to the pre-war diets with respect to the neral elements, calcium and iron, and the vitamins of the B mplex. Only with respect to vitamins A and C has there been y reason to suspect its adequacy.

etetic Priorities

the outbreak of the war it was evident that attention would ve to be given to the special dietetic needs of growing children, gnant and nursing mothers, and the sick. The nutritional tors of particular importance for these categories are first-class tein, calcium, and vitamins A and C, a deficiency of which stituted a potential danger in the war-time diets. To prevent e development or minimize the effects of such deficiencies, the vernment not merely undertook a campaign of increased pro- ction, as outlined above, but introduced the measures set out ow, which gave a priority supply of food and vitamin supple- nts to those whose needs were greatest.

a) Special allowances of vitamin A (as cod-liver oil or halibut r capsules), vitamin C (as orange juice, rose-hip syrup and ck-currant juice), and milk to pregnant and nursing mothers, ants and children under 5.

(*b*) An extra half meat ration to expectant and nursing mothers.

(*c*) Milk and meals in school for children of school age.

(*d*) The setting up of industrial canteens and British Restaurants where workers could obtain meals cheaply and in addition to the ration.

(*e*) The provision of extra cheese rations to agricultural workers and miners who were unable to obtain meals at canteens.

(*f*) Extra rations and special priorities to invalids and persons on special diets, thus:

- (1) double meat and fat ration and extra cheese and milk ration to diabetics;
- (2) special milk allowance to patients with gastric disorders and those suffering from tuberculosis;
- (3) extra meat rations to patients suffering from steatorrhœa and hepatitis;
- (4) special priorities of milk, eggs and fish to hospitals, etc.

(*g*) Compulsory fortification of foodstuffs, namely:

- (1) addition of vitamins A and D to margarine;
- (2) introduction of calcium into National flour.

Effects of Five Years of War-time Diet on National Health

1. *General Economic Efficiency.* Despite the housing, transport and black-out difficulties, the war-time productivity per head of population in Great Britain is stated to have been the highest in the world.

2. *Height, Weight and Growth of Children.* The consensus of opinion among school-teachers, social workers, school medical officers and research workers who carried out nutritional surveys, is that the health of the children of Britain remained satisfactory right through the war. A recent assessment of the nutritional state of nine-year-old school-children in certain districts in Scotland by School Medical Officers of wide experience indicated that from 96 to 98·5 per cent of the children examined were in a satisfactory or specially good state of nutrition.

War-time conditions have made it difficult to obtain statistical data based on accurate measurements by means of which the pre-war and post-war child of similar age and social circumstances can be compared. The *Annual Report on Medical Inspection and Treatment of School Children* for the year ending July 31st, 1943, issued by the Corporation of Glasgow, gives the following information on the increase in height and weight of children investigated in the same way and in the same district of the city in 1943 as compared with that before the war.

Children 5 *years of age*

Boys: increase 0·40 in. and 1·24 lb.
Girls: increase 0·28 in. and 0·97 lb.

Children 13 *years of age*

Boys: increase 0·75 in. and 2·90 lb.
Girls: increase 0·72 in. and 2·94 lb.

The Parliamentary Secretary to the Ministry of Food stated in August 1944 that a recent investigation of some 30,000 adolescent and adult males revealed no evidence of unsatisfactory growth or loss of weight. In addition, he said there was reason to believe that the war-time diet had done much to banish the 'middle-aged spread' which had been so common in males between the ages of 35 and 55.

This highly satisfactory state of affairs is all the more significant in view of certain evidence that during 1941–2, when the amount of food reaching the consumer was at its lowest level, a deterioration in the nation's nutritional status had begun. Thus, Milligan's (1942) surveys of the health of elementary school-children in 1941–2 showed a reduction in the average gain in height and weight and a lowering in the powers of endurance as compared with those shown in pre-war tests. His findings for 1942 and 1943 showed a progressive improvement. Similarly, MacMillan and Inglis (1944) reported that the number of cases of manifest scurvy treated in the hospitals of Edinburgh and Glasgow showed a significant increase in 1941–2, but had already begun to recede by 1942–3.

3. *Infectious Diseases.* Despite the dangers of overcrowding and reduced ventilation due to the black-out, shelter life, etc., the incidence of the majority of the common infectious diseases, with the exception of venereal diseases and tuberculosis, was actually less in 1944 than before the war. Notifications in Scotland for diphtheria, scarlet fever, measles, whooping-cough, typhoid and paratyphoid fever were fewer in 1943 than in 1938; and the incidence of cerebrospinal meningitis was 55 per cent lower than in 1940. The venereal diseases are, of course, social, not nutritional, and the increase in tuberculosis was due at least as much to the unhygienic conditions mentioned above as to any defects in the diet. It should be noted that the death rate for tuberculosis has steadily fallen since 1941 and reached a record low level in 1945.

4. *Infantile and Maternal Mortality.* Two of the best indices of the country's nutritional state are the rates of infantile and

maternal mortality. Despite increasing difficulties with housing and the black-out, and shortage of nurses, doctors and hospital accommodation, these have shown an encouraging decline. The infant mortality figure of 56 per 1,000 live births for 1945 was the lowest in Scotland's annals, the previous lowest having been 65 in 1943 and 1944. Since 1939 stillbirths in Scotland have been reduced by 9 per 1,000 of the population. The neonatal death rate fell from 36·5 per 1,000 live births in 1939 to 29 in 1945. The maternal death rate for 1945 was 2·9 per 1,000 live births, the lowest ever recorded in Scotland. A similar improvement in the mortality rates was shown in the vital statistics for England and Wales.

5. *Incidence of Nutritional Deficiency Diseases.* Research workers, social workers and experienced clinicians alike are all agreed that frank deficiency diseases such as scurvy, beri-beri and pellagra are as rare to-day in Great Britain as they were in pre-war days. When such diseases are seen, in the majority of cases it is found that the patient is suffering from mental or gastro-intestinal disease, or is an elderly person, usually unemployed, or living alone on a small pension in a lodging house and cooking and preparing his own meals. The number of cases of scurvy treated in the municipal and voluntary hospitals of Edinburgh and Glasgow in 1941–2, when the nation's diet was at its worst, was only 111 (MacMillan and Inglis, 1944). Sub-clinical states of vitamin deficiency, as determined by laboratory tests, would appear to be not uncommon, though probably no commoner than before the war; and in any case, the significance of this method of assaying is still open to question.

Large-scale surveys of the incidence of anæmia, as carried out by one of the authors (L. S. P. D.) as well as by others in Great Britain, show that, in general, the position with respect to iron deficiency anæmia is satisfactory. Anæmia, often mild in degree, is still common in certain groups, *e.g.* in infants, and in women, both pregnant and non-pregnant, but its degree and frequency are certainly no greater (and are possibly less) than before the war Consecutive surveys carried out in Edinburgh under the direction of the author in 1942, 1943 and 1944 showed progressive improvement in the hæmoglobin levels of school-children and pregnant women, and this was attributed mainly to the increased intake o iron resulting from the introduction of National wholemeal flour in 1942.

6. *Rickets.* The lack of uniformity in the age groups studied, and

of the criteria on which the diagnosis of rickets was based, make it difficult to compare previous surveys with the 1944 survey carried out by the British Pædiatric Association on behalf of the Ministry of Health. Depending on whether the diagnosis is based on clinical, radiological or biochemical evidence, the incidence of rickets will be found to vary. All are agreed, however, that 'florid' rickets is as rare to-day as it was in the immediate pre-war period. Even when a radiological examination was accepted as the basis for diagnosis, only 106 children out of 5,283 investigated in 23 centres throughout the country showed signs of active rickets in 1943.

7. *Dentition.* The researches by M. Mellanby during the past twenty-five years have clearly established the prominent part played by certain nutritional factors in determining the structure of the dental tissues and their resistance to disease (see p. 11). Efforts to improve the nutrition of pregnant women, infants and young children, which had been proceeding for many years, were accelerated by the Government's war-time nutritional policy. The success of this policy was clearly indicated by the survey carried out by M. Mellanby and H. Coumoulous on London County Council school-children in 1943. A summary of this work is given on p. 11.

It should be remembered that some considerable time must elapse before the full benefits can be expected from the increased intake of calcium and vitamin D secured for infants, children and pregnant women by the Government's policy of priority rations. The clinical results already obtained warrant an optimistic outlook for the future.

8. *Vital Statistics.* In a number of instances, both in this chapter and elsewhere, we have quoted statistics for Scotland in preference to those for England (which, however, show a similar trend). We have done this for several reasons. The senior author has been closely connected with the inception and execution of many investigations and nutritional surveys undertaken in Scotland, and possesses a first-hand knowledge of the factors relating to the health of the Scottish people. In addition, the Scottish vital statistics are particularly illuminating, since conditions known to influence the health of the nation adversely are present to a greater degree in Scotland than in England. Thus overcrowding, with its consequent risk of cross-infection, is officially stated to be eight times worse in Scotland than in England. Despite a further deterioration in this respect due to the cessation of building during five years of war, and despite the harmful effects of black-out conditions, overwork and mental strain, a steady improvement of Scotland's vital statistics

has occurred. Accordingly, it would appear reasonable to attribute this result mainly to the improvement in the nutrition of the poorer sections of the community which has resulted from increased purchasing power and from the more even distribution of nutrients brought about by the measures discussed above.

9. *Effect on Different Sections of the Population.* The two questions asked in the opening paragraphs of this Chapter can now be answered in brief.

1. The more fortunate members of society, whose diet was formerly unrestricted by economic circumstances, have had their intake of many of the essential nutrients reduced as a result of the war; but these reductions have done no material harm. In the small proportion of people who consistently ate and drank too much, there has probably been an actual improvement in health.

2. Improved economic conditions, the more even distribution of essential foodstuffs brought about by rationing, the introduction of national flour, and the fortification of certain foodstuffs have certainly prevented any further deterioration in the diet of the less fortunate section of the population who, before the war, could not buy an adequate diet, and in certain categories at least (*e.g.* pregnant women, infants and school-children) have led to an undoubted improvement.

Conclusions

Out of the tragedy and calamity of war, certain benefits may accrue to the nation. Millions of young men and women have spent several critical years of their lives in the Forces, during which they enjoyed the benefits of fresh air, physical exercise and adequate nutrition to an extent which would have been available to only a few in their peace-time occupations in the office and the factory. The tragedies and sacrifices of all sections of the community have stirred the national conscience to such a degree that there exists a sincere and widespread determination to see that the amenities of life are more evenly distributed among all sections of the people. The Government's white papers dealing with education, housing, unemployment and medical services are an expression of this national desire for improvement of the social status of the less fortunate members of society. Lastly, war inevitably brings a great impetus to scientific research of all kinds. The success of the Government's war-time food policy can be attributed largely to the sound scientific basis on which it was founded. It

TABLE 7

ESTIMATED SUPPLIES OF NUTRIENTS AVAILABLE FOR CIVILIAN CONSUMPTION *

Pre-war to 1943

(Per head per day)

	Supplies, Pre-war	Supplies, 1943	Percentage Change 1943 compared with Pre-war
Calories	2,984	2,827	− 5
Protein, animal (grams)	43	40	− 7
vegetable (grams)	38	47	+ 23
Total (grams)	81	87	+ 7
Fat	130	113	− 13
Carbohydrate	373	366	− 2
Calcium (mg.)	694	1,054	+ 52
Iron (mg.)	13	16	+ 27
Vitamin A (I.U.)[1]	3,868	3,882	—
Ascorbic acid (vitamin C)[2] (mg.)	112	127	+ 13
Aneurin (vitamin B_1) (mg.)	1·2	1·9	+ 60
Riboflavin (mg.)	1·6	2·1	+ 30
Nicotinic acid (mg.)	18	19	+ 3

[1] One I.U. of vitamin A = 1·8 micrograms of beta carotene.

[2] The loss of vitamin C in storing, marketing, preparing and cooking fruit and vegetables will greatly reduce the figure for vitamin C actually consumed.

* This and the next Table from *Food Consumption Levels in the United States, Canada, and the United Kingdom.* London, 1944. By kind permission of H.M. Stationery Office.

TABLE 8

ESTIMATED SUPPLIES MOVING INTO CIVILIAN CONSUMPTION IN THE UNITED KINGDOM

Pre-war to 1943

(lb. per head per year)

	Supplies, Pre-war	Supplies, 1943	Percentage Change 1943 compared with Pre-war
Fluid milk	217·7	291·6	+ 34
Whole-milk cheese	8·8	11·8	+ 34
Meats	136·4	107·3	− 21
Oil and fats	45·6	38·4	− 16
Sugar and syrups	94·5	65·0	− 31
Potatoes	177·0	255·8	+ 45
Tomatoes and citrous fruits	46·8	23·2	− 50
Leafy green and yellow vegetables	99·3	132·7	+ 34
Grain products	211·0	247·4	+ 17

is to be hoped that those war-time measures which have be
shown to be of such value to the people's health, *e.g.* priori
rations of milk for infants, school-children and pregnant wome
school meals and works canteens, National wheatmeal bread, et
will be continued as part of the permanent peace-time food polic

PART IV

DIETETIC TREATMENT IN ESTABLISHED DISEASE

CHAPTER XI

DIABETES MELLITUS

The introduction of insulin in the therapy of diabetes, besides revolutionizing treatment and rendering the prognosis of the disease immeasurably more favourable, has had the added advantage of simplifying the dietetic treatment. As a result, the management of a case need no longer be considered the exclusive preserve of the specialist in dietetics. This belief is a legacy of the days when diabetic diets had to be calculated in terms not only of Calories, carbohydrates, fats and proteins, but also of ketogenic-antiketogenic ratios as well. With the introduction of insulin and diets only moderately restricted in carbohydrate, the physician no longer needs to be familiar with complicated food tables and dietetic calculations; all that is required is a simple system of diets and a working knowledge of sufficient food values and exchanges of foodstuffs to enable the diets to be modified to suit individual patients.

DIFFERENTIAL DIAGNOSIS OF GLYCOSURIA

Although it is true that the majority of patients in whom glycosu is discovered are suffering from diabetes mellitus, nevertheless existence of the non-diabetic glycosurias must be rememb While diabetes should be the condition uppermost in mind wh glycosuria is revealed, the patient should not be treated as dial until the diagnosis is established, either by the presence of defin diabetic symptoms and ketosis, or by appropriate confirmato biochemical tests. If the presence in the urine of drugs such a salicylates and substances excreted in combination with glycuronic acid, *e.g.* chloral, camphor and turpentine, can be excluded, the conditions other than diabetes mellitus in which reducing substances may be present in the urine are:

1. **Renal glycosuria** – an hereditary, familial condition in which the renal threshold for glucose is lower than the normal value

of 0·17 grams per 100 c.c. of blood, so that glucose is excreted in the urine when the blood sugar is still within normal limits. The glycosuria is usually unaccompanied by any symptoms. Rena glycosuria can be distinguished from symptomless diabetes by the glucose tolerance test.

2. Glycosuria due to the 'Lag' Type of Blood Sugar Curve. In this less common condition the blood sugar rises sharply after a carbohydrate meal to a level above the renal threshold so that glycosuria occurs, but returns to a normal level within a period of two hours. The usual explanations given for this type of blood-sugar curve are that there is either a delay in the mechanism for storage of carbohydrate, which, nevertheless, functions normally once it comes into action, or an excessively rapid absorption There is still a doubt as to whether this condition should be considered a potentially diabetic one, but most authorities now consider it to have no prognostic significance. Like renal glycosuria, it can be distinguished from true diabetes by the glucose tolerance test.

3. Glycosuria due to other Endocrine Disorders. In overactivity of the anterior pituitary, thyroid or adrenal glands glycosuria may occur, and it may, therefore, be present in such conditions as acromegaly, Cushing's syndrome, exophthalmic goitre and tumours of the adrenal glands.

4. Glycosuria of Pregnancy. The occurrence of reducing substances in the urine for the first time during pregnancy may be due to one of three causes:

(i) Renal glycosuria of pregnancy – a temporary lowering of the
enal threshold for glucose, which returns to normal just before or
ter parturition.

(ii) Diabetes of pregnancy. Occasionally a true diabetes may
e during pregnancy and disappear after delivery. The condition
ost probably due to pituitary over-activity during pregnancy

i) Lactosuria. Lactose may occur in the urine during the
months of pregnancy or during lactation, and can be distin
shed from glucose by the fermentation test. Lactosuria is of no
gnificance.

The rare condition of pentosuria, usually a familial condition
eed only be mentioned; it is of no pathological significance.

In any doubtful case the importance of distinguishing, by means of the glucose tolerance test, between true diabetes mellitus and other causes of glycosuria should be emphasized here. While it is true that a high fasting blood sugar usually signifies diabetes, the fasting blood sugar may be normal in mild diabetes or may be slightly raised as a result of excitement or nervousness. The extra trouble to the patient of submitting to the glucose tolerance test is more than compensated for by the certainty of knowing that the dietary restrictions to which he must submit for life are, in fact, essential.

The Three Grades of Diabetes

When diabetes mellitus has been established as the cause of glycosuria in a patient, an attempt should be made, before starting treatment, to assess the severity of the diabetic condition.

Lawrence and Archer (1936) have suggested that in a normal individual there is probably a constant small secretion of insulin which takes charge of the carbohydrate produced in endogenous metabolism, while each meal containing carbohydrate calls forth an added amount of insulin. On this theory Lawrence (1938) has divided diabetes into three grades of severity:

1. **Mild Cases.** The patients are usually middle-aged and often obese. They can secrete enough insulin to control the carbohydrate produced in endogenous metabolism, so that the blood sugar is normal during fasting, but are unable to secrete sufficient for the metabolism of the carbohydrate taken in the meals. Hyperglycæmia and glycosuria therefore result.

These patients can utilize sufficient carbohydrate to prevent the onset of ketosis, unless some complication, such as an infection or gangrene, reduces the efficacy of their insulin.

2. **Moderate Cases.** This group comprises the patients whose basal insulin secretion is somewhat reduced and is no longer sufficient to utilize all the carbohydrate produced in endogenous metabolism, so that the fasting blood sugar level is raised. The main hyperglycæmia, however, again occurs after meals, but there is enough natural insulin secretion left to ensure that a completely diabetic condition does not supervene when the effect of injected insulin has worn off.

Diabetes developing in children and young adults is usually of this type, and there is a tendency for the disease to progress to the third group, and then remain stationary.

3. **Severe Cases.** Patients belonging to this group may be considered as having no insulin secretion from the pancreas at all, and are what Lawrence calls 'absolute diabetics'. Such patients are entirely dependent on injected insulin, and as soon as the effect of a dose wears off they relapse into a completely diabetic condition with marked hyperglycæmia, glycosuria and ketosis.

Assessment of Severity of Disease

Although in some cases a careful study of the effect of treatn on the course of the disease may be required before the patient be finally placed in one of these somewhat arbitrary group tentative assessment of the severity of the diabetic condition usually be made from the following factors:

1. The age of the patient. Diabetes developing in elderly in duals is usually of the mild type. In children and young ad the disease begins either as the moderate or severe type.

2. The severity of the diabetic symptoms, such as th polyuria, loss of weight, weakness.

3. Physical examination of the patient may be essent negative, but may reveal evidence of marked loss of weight dehydration, or the presence of any of the complications of diab such as neuritis, carbuncle, cataract, retinitis or gangrene.

4. Biochemical investigations:
   - (i) Urine examination – glucose and ketone bodies.
   - (ii) Fasting blood sugar level.
   - (iii) Glucose tolerance test.
   - (iv) Alkali reserve of the blood.

Patients who can be assigned to the group of mild diabetic elderly persons, usually obese, with few or no diabetic sympt and no complications, whose glycosuria is often discovered du routine examination for some non-diabetic complaint. The gl suria is not accompanied by ketonuria, and if biochemical test carried out, the fasting blood sugar will be found to be w normal limits, while the blood sugar curve shows only a moder excessive elevation and is returning to, although not reachi normal level at the end of two hours. It is only in this grou patients that treatment by diet alone without the use of insul likely to be successful.

Cases not conforming to the criteria given above are e moderate or severe and call for insulin as well as dietetic treatn

GENERAL PRINCIPLES OF TREATME

The question of dietetic treatment in diabetes is so interwoven insulin therapy that the two must be considered together if clear picture of the rational lines of treatment is to be draw

Before considering the treatment of the individual case, convenient to discuss some general points which are applica all cases.

1. The Caloric Value of the Diet. Before the introduction of insulin, an effort was made by dietetic restrictions to keep diabetic patients about 10 per cent under the ideal weight corresponding to their age, height and sex. This reduced the basal metabolism slightly and, therefore, lessened the food intake required to maintain equilibrium.

Such rigid restriction is no longer necessary, as an adequate diet can now be balanced by insulin injections. This does not mean, however, that an unlimited Calorie intake is permissible. Most normal people eat more than is needed to fulfil the requirements imposed by the basal metabolism and the bodily activities, the excess food being burned up by what is termed 'luxus consumption' (see p. 348). The 'luxus consumption' of carbohydrate requires the presence of insulin, and the Calorie intake appropriate for a diabetic will, therefore, be the minimum amount which allows him to maintain his normal weight and carry out his daily activities with the normal sense of well-being. The Calorie intake appropriate for adult male diabetics is usually between 1,800 and 2,500 alories, depending on the amount of muscular work performed, while female diabetics are generally found to maintain weight and health on diets of between 1,600 and 2,200 Calories.

Obesity is known to increase the severity of diabetes, and a duction in the weight of an obese diabetic may be accompanied y an improvement in carbohydrate tolerance. For both these asons, it is of the greatest importance to attempt to reduce the ight of obese diabetic patients to as near their ideal weight as sible by the use of diets containing as little as 1,200 Calories ee p. 240).

2. The Restriction of the Carbohydrate Intake. While some striction of carbohydrate is necessary in every case, this does not ve to be carried to the extremes unavoidable in the diets escribed for diabetics before the introduction of insulin. In pre- ulin days, diets contained as little as 50 to 70 grams of carbo- rate, but much more liberal allowances are now made, since re is no possibility of imposing a strain on the patient's damaged creatic islets if the carbohydrate allowance is properly balanced injected insulin. In fact, in diabetics as well as in normal dividuals the carbohydrate tolerance is lower on a very low rbohydrate intake than on a more liberal one, and this applies th to endogenous insulin secretion and injected insulin, so that a derate increase in carbohydrate intake does not necessarily an a corresponding increase in the insulin requirement.

In some clinics, the pendulum has swung considerably so th diets containing as much as 250 to 300 grams of carbohydrate a prescribed, but it is probably better to restrict the carbohydra to 120 to 200 grams daily, as insulin dosage is more easily adjust and maintained at this intermediate level of carbohydrate intak In patients treated by diet alone, carbohydrate restriction ma have to be carried a little further in order to keep the urine suga free, but the diet should not contain less than about 90 grams carbohydrate daily. If glycosuria occurs on this intake, the patie must be treated with insulin.

The increase in the carbohydrate quota allowed when insul is used has made diets easier for the doctor to prescribe and mo pleasant for the patients to take than in the days of the u appetizing and often nauseating low carbohydrate-high fat diet This is an important factor, as patients are more willing to keep the higher carbohydrate diets, which are more economical a approximate more closely to a normal diet.

3. The Restriction of the Protein Intake. Protein by i specific dynamic action increases metabolism. This is undesira in a diabetic, when an effort is being made to keep the Calo intake at the minimal level necessary for maintenance of weig and well-being. It must also be remembered that a little over per cent of the protein of the food can be converted into gluco so that an excessive protein intake may upset the balance of a d on which the patient had been stabilized. Therefore, a modera restriction of the protein intake should be made when prescribi a diabetic diet. The suggested intake is about 70 to 85 grams persons weighing between 10 to 12 stone, and is well above t amount necessary to maintain nitrogenous equilibrium. Th calculation is based on the well-recognized fact that an intake 0·5 gram of protein per pound of ideal body weight is a satisfacto amount. A moderate variation above or below this intake is of great importance, but we do not advocate the system suggested some of allowing diabetics to eat as much protein and fat as th like, provided that the carbohydrate foods are weighed.

4. The Maintenance of a Safe Ketogenic : Antiketoge Ratio. It was formerly believed that the complete oxidation fatty acid required the simultaneous oxidation of carbohydra the ketone bodies 'burning in the flame of carbohydrate'. Rece experimental observations suggest that the following explanat is more likely to be correct. Fatty acids are normally broken dow independently of carbohydrate, to carbon dioxide and water way of hydroxybutyric and acetoacetic acids. The body can only deal with a limited amount of these substances. The quantity ordinarily produced is well below this limit, and ketone bodies therefore do not accumulate. When, for any reason, the amount of carbohydrate oxidized is greatly reduced (whether from insufficient intake or failure in utilization), the body is forced to obtain an unduly high proportion of its energy requirements from the combustion of fats. Accordingly, there is increased production of ketone bodies with which the body is unable to deal, so that these substances accumulate and appear in the urine. This sets a limit to the restriction of carbohydrate in the diabetic diet. Before the introduction of insulin, when the carbohydrate of the diet had to be reduced to the lowest limit of safety, the ratio of available glucose to fatty acid had to be calculated to ensure that it was not less than 1 to 1·5, the critical level below which the oxidation of fat is incomplete and ketosis sets in.

Such calculations, the bugbear of the general practitioner, are no longer necessary since the amounts of carbohydrate prescribed in diabetic diets at the present time – 90 to 200 grams – ensure that the glucose : fatty acid ratio is well above the critical level, and, therefore, that severe ketosis cannot occur in a properly treated diabetic except in the following circumstances:

(i) When the patient disobeys the dietary regulations, or omits insulin if this has been prescribed.

(ii) When infection diminishes the activity of insulin, whether the latter is secreted by the patient's pancreas or injected.

5. The Calculation and Arrangement of the Diet. The physician will have to decide how many Calories the patient will probably require, how much carbohydrate he should be given, and what the protein intake should be. This latter can be calculated as 0·5 gram per pound of ideal body weight (see Table 16 for average body weights according to age, height and sex).

The caloric value of a gram of either protein or carbohydrate is approximately four, so that the Calories derived from these two classes of foodstuff can be calculated by multiplying the number of grams of each in the diet by four. If the Calories derived from carbohydrate and protein are subtracted from the total Calories required, the remainder represents the Calories to be supplied by fat, and this figure divided by nine (the number of Calories derived from the combustion of 1 gram of fat) gives the number of grams of fat required. As an example, let us suppose that it is decided to give a male diabetic patient, whose ideal weight is 10 stone, a diet of 2,000 Calories, with a carbohydrate quota of 150 grams. The protein quota will be 140 (*i.e.* 140 lb.) × 0·5 = 70 grams

The Calories derived from protein = 70 × 4 = 280
„ „ „ „ carbohydrate = 150 × 4 = 600

Therefore the Calories to be derived from fat will be 2,000 less (280 + 600) = 1,120 Calories. Since 1 gram of fat yields 9 Calories the diet will have to contain $\frac{1120}{9}$ = approximately 124 grams of fat.

A diet prescribed in terms of grams of protein, fat and carbohydrate must now be translated into understandable dietary instructions for the patient. This is a somewhat laborious proces for one not trained in dietetics, and to simplify matters we hav given examples of diabetic diets with various carbohydrate contents and caloric values (see pp. 448 to 462). These diets, it i hoped, supply most of the types which the physician will need but when any of them require to be modified by a decrease o increase in Calories, carbohydrate, protein or fat, this can easily b carried out by the use of Table 21 (p. 443), which gives 100 Calori portions of different foodstuffs, arranged in three groups accordin to whether they yield predominantly carbohydrate (20 to 2 grams), protein (approx. 15 to 16 grams) or fat (approx. 12 grams) Monotony is a potent stimulus to a patient to break his diet Hence an important point in the prescription of the diet, an this applies to any diet whether used in the treatment of diabete or any other disease, is the provision of exchanges for many of th articles of the diet, these exchanges consisting of foodstuffs o approximately equal composition and caloric value. Lists of exchanges for the meat, fish, eggs, bread, fruit and vegetables ar given in Tables 19, 20 and 25. The patient must be expressl warned not to partake of the following foods: sugar in any form (*e.g.* icing on cake, sweets, etc.), tinned fruit or fruit bottled wit sugar, sweetened mineral waters, or jam (except if allowed in h diet). If the exclusion of sugar is trying to the patient, sweetenin by the use of saccharin is permissible. A ½ grain saccharin tabl is enough to sweeten a large cup of tea, or a serving of custar or milk jelly. In cooked dishes, saccharin should, if possibl not be added until a few minutes before the food is ready to b served.

6. The Education of the Patient. In no disease is the intelligent co-operation of the patient or a relative more important f success in treatment than in diabetes. The nature of his disea must be explained in simple terms to the patient and the reasons given why the doctor's instructions must be scrupulously adhered to. The dangers incurred if these instructions are not carried out must also be mentioned, the emphasis to be laid on this latter point varying, of course, with the mentality of the patient.

There is no doubt that it is easier to carry out the initial stabilization of the diabetic patient as regards diet and insulin in a hospital or nursing home, and the education of the patient in the part he is to play in treating his disease is also more readily undertaken in such an institution. Whether or not it is possible to arrange for the patient's hospitalization, he must be taught the following points during the process of stabilization:

(i) To test his urine for sugar. A rough quantitative idea of the amount of sugar in the urine can be obtained from the colour of the precipitate, and for this purpose Benedict's reagent is to be preferred to Fehling's solution. The test should be carried out as follows (Bray, 1938):

Place 5 c.c. Benedict's qualitative reagent in a test tube, add 5 drops of urine (or 0·25 c.c.), mix and place in boiling water-bath 5 minutes or boil over flame 1 to 2 minutes. A green, yellow or red colour, with a yellow or red precipitate is positive. A clear or cloudy blue colour with a greyish precipitate is negative. Allow to cool at room temperature before discarding as negative. A rough estimation of the amount of sugar in the urine can be made by judging the colour – *e.g.* Red = 3 per cent; Yellow = 2 per cent; Greenish-yellow = 1 per cent; Green = 0·5 per cent; Blue = no sugar.

(ii) To weigh the different articles of food in his diet, and to use lists of food exchanges in making up different menus. The patient must provide himself with scales and get into the habit of weighing the prescribed diet accurately. At the beginning of treatment every article of the diet should be weighed, but after some weeks or months dependable and intelligent patients will have acquired considerable accuracy in estimating the weight of foodstuffs by eye. When this point in training is reached the rules can be relaxed somewhat, and the patient made to weigh only the articles of food rich in carbohydrate. When the patient has achieved this stage, he may be encouraged to take occasional meals away from home, so that he ceases to look on himself as an invalid.

To keep his eye trained, however, the diabetic should practise weighing every article of food eaten during one week at intervals of a few months.

(iii) Either the patient or a relative must be taught how to give insulin injections. He must be shown how to sterilize syringe, needle, cap of insulin phial and skin, and how to give injections into the subcutaneous tissue and not into the skin or muscle. He must also learn how to distribute the injections over various sites on the body so as to prevent fat atrophy or induration of the subcutaneous tissues.

(iv) The patient must be told the symptoms of hypoglycæmia and must be warned to be on the look-out for the early symptoms, especially after unwonted exercise. The rapid response of th symptoms to the consumption of sugar must be pointed out, an every diabetic being treated with insulin must carry with him wherever he goes some sugar-containing food such as lump sugar chocolate or sweets. He should be told to take 10 grams of carbohydrate as soon as hypoglycæmic symptoms are recognized, an another 10 grams fifteen minutes later if the symptoms have no disappeared. The following amounts of different substances contain roughly 10 grams of carbohydrate: two lumps of sugar, one third of a 2d. bar of chocolate, two heaped teaspoonfuls of glucos or Ovaltine, or eight tablespoonfuls of orange juice.

(v) The proper hygiene of the feet must be insisted on, especiall in elderly diabetics, in order that the danger of diabetic gangren may be minimized. If possible, corns and callosities should b treated only by a competent chiropodist who has been informe of the diagnosis. The toe-nails must be kept short. The feet shoul be washed once daily, dried carefully, rubbed with methylate spirits and powdered. Footwear must be comfortable and room

Cuts and blisters in any situation must be attended to carefull and kept clean.

(vi) Any infection, even a mild one, is a major illness for diabetic, and without alarming the patient this point should b explained to him. He should be instructed to go to bed and call i his doctor if he develops any infection, even a mild one such as cold or tonsillitis. Likewise, if for any reason such as a gastr intestinal upset the patient cannot take his diet, he must b instructed to take the insulin prescribed for him plus the carb hydrate of his diet or its equivalent in the form of fruit juic sugar, etc., and send for the doctor. The failure to continue insul therapy in the presence of infections or gastro-intestinal upset i in our experience, the most common cause of diabetic coma.

ADJUSTMENT OF TREATMENT TO SEVERITY OF DISEASE

In the above sections, the grades of severity into which diabetes can be divided and the more general aspects of dietetic treatment were discussed; the treatment of the individual patient under different conditions can now be described. The criteria mentioned on p. 232 will indicate in the majority of cases whether the disease can be considered mild, moderate or severe and whether insulin will or will not be required.

1. Treatment of Mild Cases

The patient is usually elderly, with few or no diabetic symptoms, no complications of diabetes and with only glucose and no ketone bodies in the urine.

The first step in treatment is to get the patient's urine free from sugar. This is best done by giving him a diet low in Calories and with a correspondingly small carbohydrate quota. A sub-basal diet containing as little as 1,200 Calories should be given. If the patient cannot maintain his normal activities on this intake, he can be kept in bed during this first stage of treatment. The diet, Diet No. 1 (p. 448), should contain about 50 grams of protein, 85 grams of fat and 65 grams of carbohydrate. The patient should be kept on this diet for not more than seven to ten days, and if the 24-hour specimen becomes sugar-free within that period, he is ready for the next stage in the treatment. If sugar is still present, the case cannot be considered so mild as not to need insulin. It is important that the response of a mild case being treated by diet alone should be judged from the results of the tests on the 24-hour specimen of urine. If random samples of urine are taken for testing, it is possible that one, passed several hours after a meal, may be sugar-free while another, passed shortly after the next meal, may contain sugar in considerable amounts. Examination of the 24-hour urine specimen will prevent such intermittent glycosuria being missed.

The patient's urine having become sugar-free on the sub-basal diet, an increase in the diet is made up to the level of the patient's basal requirements, say about 1,500 Calories. The diet, Diet No. 2 (p. 450), should contain about 55 grams of protein, 110 grams of fat and 80 grams of carbohydrate.

If, after a week or so on the basal diet, the patient's urine remains sugar-free, a final increase in the diet can be made, so as to meet the patient's maintenance requirements. As the patient is usually an elderly person, the Calorie intake need not be a libera one, and about 1,800 to 2,000 Calories is usually sufficient t maintain the patient's weight and strength. The protein allowanc should be about 80 grams, the fat 130 grams, and the carbohydrate 90 to 100 grams (Diet No. 3, p. 451). In very mild case it may be possible to increase the carbohydrate quota to a littl above 100 grams without causing a return of the glycosuria.

Provided the patient's urine remains sugar-free and he maintain his weight, the disease may be considered to be under control. I the weight and strength are not maintained on an intake of 1,80 Calories, the diet can be increased by adding one or more 10 Calorie portions of fat (see p. 443). Actually, eight such portion could be added before the glucose : fatty acid ratio reached th critical level, but this would bring the diet up to 2,600 Calories, a intake which an elderly individual, otherwise healthy, will neve need to maintain his weight.

If sugar appears consistently in the urine at any of the thre stages mentioned (sub-basal, basal or maintenance diet), th patient cannot be considered to have only mild diabetes and mus be treated with insulin. It is quite frequently found, howeve particularly in obese patients, that after a time the dose of insuli can be reduced and in some cases dispensed with, the patien having recovered sufficient of his lost carbohydrate tolerance t be classed now as a mild diabetic.

It is doubtless true that a greater percentage of elderly patien could be kept sugar-free without insulin if the carbohydrate quo was lower than 90 grams. We consider 90 grams to be the mir mum amount desirable in a maintenance diet for a mild case, f the reasons that a diet containing much less than this quantity approaching the limit of safety of the glucose : fatty acid rati and also because patients have less vigour on such a diet whi they are tempted to break on account of its unpalatability. Even restriction to 90 grams of carbohydrate imposes a certain hardsh on many patients, and in such cases the patient must deci whether this restriction is preferable to the use of insulin with more liberal carbohydrate intake.

Obesity in Diabetes. Many of the mild cases are obese; eve effort should be made to reduce the weight towards a normal lev since, as was stated already, obesity tends to make diabetes wor The diet prescribed for such patients should, therefore, be bel their maintenance requirements – either 1,200 or 1,500 Calor depending on the degree of obesity. The carbohydrate quota these low Calorie diets, however, should not fall below 90 to 100 grams. This obviates the risk of ketosis which may occur when the patient burns up his body fat to make up for the deficient energy intake. If the butter ration in Diet No. 3 is reduced from 3 oz. to 1½ oz., the caloric value is reduced from 1,850 to 1,500 without affecting the protein or carbohydrate quotas. For patients markedly overweight the diet should contain approximately 1,200 Calories and 100 grams of carbohydrate. Diet No. 5 (p. 454) will meet these requirements. Reduction in weight often increases the carbohydrate tolerance in diabetes, and obese patients, who required insulin when overweight, may be able to do without it when their weight has been reduced.

2. Treatment of Moderate and Severe Cases

While most authorities are agreed that the recently introduced, slowly acting type of insulin, zinc protamine insulin, is a definite advance on the ordinary soluble insulin in the treatment of diabetes, the new compound, like the old form of insulin, has certain disadvantages. The best idea of the relative merits of the two forms of insulin is gained by comparing how far each of them can imitate the natural secretion of the pancreas. As has already been stated, normal individuals secrete a constant small amount of insulin to deal with the sugar produced in endogenous metabolism, while the carbohydrate absorbed from each meal is acted on by insulin which is rapidly secreted.

Ordinary Insulin. When ordinary (soluble) insulin is injected subcutaneously before a meal, it produces a rapid fall in blood sugar, the lowest value being reached two to four hours after injection; the effect then dies away, usually about six to eight hours after injection. Ordinary insulin, therefore, imitates the increased secretion of insulin occurring in normal persons after a meal. If, however, a diabetic patient is being treated with two injections of ordinary insulin daily, he will have to depend on his own subnormal supply of insulin for almost half the 24-hour period, so that he may be verging on hypoglycæmia after his insulin injections, but may show marked hyperglycæmia, glycosuria, and even ketosis in severe cases, before his next injection. Even when three injections are given throughout the day, the patient will still be without the benefit of injected insulin during the greater part of the night.

Zinc Protamine Insulin. This substance is actually a compound between a simple protein, protamine, and insulin, with traces of zinc added to maintain the stability of the compound an prolong its action. As prepared, zinc protamine insulin is in th form of a cloudy suspension of particles of the compound. Whe these particles are lodged in the subcutaneous tissues by injectio the compound is slowly dissolved in the body fluid and the release insulin carried away in the blood stream. Zinc protamine insul therefore imitates the constant secretion of insulin by the norm pancreas, but cannot imitate the increased insulin secretion response to meals.

Zinc protamine insulin begins to show an effect on the bloo sugar about three hours after injection. The duration of its actio depends, of course, on the dose given, and it has been found th small doses of 10 to 20 units act for about twelve hours, wh larger doses, 30 units or over, act for about twenty-four hours. T fall in blood sugar caused by zinc protamine insulin is much mo gradual than that due to ordinary insulin, so that the fluctuatio between hyperglycæmia and hypoglycæmia occurring when or nary insulin is used are reduced.

The prolonged action of zinc protamine insulin has several d advantages, however, which must be borne in mind when a sche of treatment is being arranged.

(i) It does not enter the circulation rapidly enough to deal wi the sugar absorbed from a meal containing more than about grams of carbohydrate.

(ii) Hypoglycæmia is much more insidious in its onset than w ordinary insulin. The subjective symptoms are much milder th those produced by an overdose of ordinary insulin, and the patie may notice only drowsiness, fatigue, headache, or slight naus with relief from these symptoms when a meal is taken. These m symptoms may, however, lead without further warning to a sev hypoglycæmic reaction, with convulsions or coma. The time onset of hypoglycæmia is not so constant as with ordinary insul and may be at any time of the day or night. The interval betw injection and reaction tends to increase with the dose, howev from about eight to twelve hours with 20 units to sixteen twenty-four hours with 40 units or over.

Another disturbing feature of the hypoglycæmia due to z protamine insulin is that the initial recovery following the gestion of sugar may be followed by a relapse owing to continued action of the zinc protamine insulin. Therefore, in treatment of hypoglycæmia resulting from an overdose of z protamine insulin, carbohydrate should be given in liberal amou such as three to four lumps or teaspoonfuls of cane sugar (approximately 15 to 20 grams of carbohydrate), and this dose should be repeated if the symptoms have not passed off within fifteen minutes.

(iii) Since zinc protamine insulin does not begin to reduce the blood sugar until about three hours after the morning injection, in severe cases there will be insufficient insulin available to control the carbohydrate taken during the first part of the day.

(iv) Zinc protamine insulin is more irregular in its action from day to day than ordinary insulin, and a patient who has apparently been satisfactorily controlled may suddenly show glycosuria for a day or two, although conditions as regards injection and diet have remained unaltered. This irregularity is probably due to occasional differences in the rate of absorption from the subcutaneous tissues.

Globin-Insulin. A third type of insulin has recently been introduced to clinical practice. The name globin-insulin has been given to a preparation of insulin combined with globin, a protein obtained from erythrocytes. Traces of zinc are added. It is said to produce its maximal effect four to eight hours after injection, and to have an action which lasts up to eighteen hours. It is also said to combine the advantages of soluble insulin and zinc protamine insulin, its action simulating the action of a mixture of both preparations. The dangers of hypoglycæmic reactions occurring during the night and the tendency to local reactions are less than with zinc protamine insulin. Although the time of onset of hypoglycæmic reactions varies in different individuals, in our experience the most usual time is shortly before the mid-day meal. Although the introduction of globin-insulin is too recent to justify a final assessment of its rôle in the treatment of diabetes, sufficient experience has already been gained to suggest that its use represents a definite advance in the treatment of selected cases.

Since the advantages of the slower-acting insulins outweigh their disadvantages, it is recommended that every new case of diabetes requiring insulin should be treated with zinc protamine insulin or globin-insulin along the lines described below. The only exceptions to this rule are patients who lead irregular lives as regards meal hours, such as gamekeepers, in whom the danger of hypoglycæmia resulting from failure to take meals at the prescribed times more than offsets the advantages of the single daily injection.

The question whether patients who have been treated with ordinary insulin should be changed over to zinc protamine insulin or globin-insulin is a difficult one. Patients who have been sat factorily stabilized on two injections of ordinary insulin daily, a to whom the routine of the injection twice daily is not irkson should not have their treatment changed unless they express preference for a single injection. Severe cases, in which t diabetes is very imperfectly controlled, with two or even th large doses of ordinary insulin daily, will do better on zi protamine insulin combined, if necessary, with ordinary insul as described below. Diabetes of mild to moderate sever is frequently well controlled by a single injection of glob insulin.

Since soluble insulin is advocated by some physicians for m cases which can be controlled with one injection, and by oth for preliminary stabilization before transferring to zinc protam insulin or globin-insulin, the following sections on the arran ments of diets and treatment in cases requiring insulin inclu schemes for both ordinary insulin and zinc protamine insulin, w suitable modifications when globin-insulin is used.

Treatment of Moderate and Severe Cases with Ordina (Soluble) Insulin

When the treatment is started, the patient will have mar glycosuria and possibly ketonuria as well. When continu supervision is possible, it is quite justifiable to place him o maintenance diet from the first, and increase the insulin dos rapidly to bring the diabetic condition quickly under control. the patient is being treated at home, however, it is probably bet to place him at first on a basal diet with a lower carbohydr quota than it is intended to give in the permanent diet. T second method will allow quick control of the diabetes with sma doses of insulin, and the diet can later be increased both in Calo and carbohydrate with the necessary small additions to the ins dosage.

Since insulin is to be given twice daily, before breakfast a before the evening meal, both the basal and the maintenance should have one-third of the daily carbohydrate quota masse each of these meals, the remaining third being distributed betw the mid-day meal and supper.

The basal diet should contain 1,500 to 1,600 Calories with ab 120 grams of carbohydrate, Diet No. 6 (p. 456). If the case i moderate one and shows no ketosis, the diet may be given al for a few days, as a reduction of the degree of glycosuria usually result from this treatment and thus enable the requisite dose of insulin to be judged more accurately.

Insulin injections are given thirty minutes before the morning and evening meals, beginning with 5 to 15 units, the initial dose depending on the degree of glycosuria. For ease and safety in stepping up the insulin doses, the examination of samples of urine passed throughout the day will prove of great value. The four samples which should be tested are:

1. Pre-breakfast, the urine passed on rising being discarded or tested separately, as it represents urine secreted during the night.
2. Before the mid-day meal.
3. Before the evening meal.
4. Last thing at night before retiring.

These urine samples give an indication of the height of the blood sugar:

(i) At the times when the effect of injected insulin is greatest, *i.e.* before the mid-day meal, some four or five hours after the morning insulin dose (sample 2), and last thing at night, about four hours after the evening dose (sample 4). If either sample 2 or 4 contains sugar, it is safe to increase the insulin dosage at the morning or evening injection as the case may be.

(ii) At the times when injected insulin is having little or no effect (samples 1 and 3).

If the patient is taught to test his own urine at the beginning of treatment, he can keep a chart of the results of these urine tests for his doctor's information, and so save the latter's time for more urgent duties. Such a chart will enable the doctor to decide whether the morning or evening dose of insulin, or both, require to be increased. Thus, glycosuria in sample 2 will be an indication for an increase in the morning dose, while glycosuria in sample 4 will show that the evening dose is insufficient. According to the results of the urine tests, the doses of insulin are increased on alternate days by 2 to 5 units, until, in moderate cases, all the urine samples are sugar-free or show only a trace of sugar. In severe cases, a sugar-free urine throughout the day is unattainable without producing hypoglycæmia at some period, owing to the rapid but short-lived action of ordinary insulin; the best that can be hoped for is that the mid-day and late evening urine samples will be sugar-free.

The patient is now ready to have his diet raised to the maintenance level, unless he is overweight, when the basal diet should be continued until satisfactory weight reduction has been attained.

A diet of about 2,000 Calories is adequate for the average patie unless he has to undertake heavy muscular work, when 2,500 even 3,000 Calories may have to be given. For a patient wh much underweight, a diet of similar high caloric value should given until the ideal weight is approached or reached. The car hydrate content of the maintenance diet may be anything fr 150 to 200 grams daily, but it will be found easier to stabilize patient on the lower of these values. A suitable maintenance d of 2,000 Calories, containing 150 grams of carbohydrate, is gi in Diet No. 7 (p. 458).

When the patient is transferred to the maintenance diet, glycosuria, previously more or less controlled on the basal d will recur or grow worse. The insulin dosage which was suffic while the basal diet was being given will, therefore, have to increased progressively, according to the results of the urine te until the four samples are again sugar-free.

As on the basal diet, a sugar-free urine throughout the day i impossible achievement in a severe diabetic, without produ hypoglycæmia during the forenoon and late evening. In s cases, insulin may have to be given three times daily, an e injection being introduced before the mid-day meal. This is ad to the inconvenience already experienced by the patient, and with three injections of insulin, the urine passed first thing in morning and before breakfast may show sugar and possibly ke bodies as well. Another method of modifying treatment f severe case of diabetes is the introduction of 'buffer' meals at time of expected hypoglycæmia, that is in the middle of the noon and in the late evening; some of the carbohydrate breakfast and the evening meal is transferred to these 'bu meals, and this may allow larger doses of insulin to be given b breakfast and the evening meal without the productio hypoglycæmia.

Treatment of Moderate and Severe Cases with Zin Protamine Insulin

The disadvantages of zinc protamine insulin which were menti on p. 242 make it necessary to modify the arrangement of th somewhat if the case is to be treated with the new insulin. I be remembered that zinc protamine insulin cannot deal wit sugar absorbed from a meal containing more than 40 gra carbohydrate, and that hypoglycæmia occurring at any ho the day and night has to be guarded against. Both these advantages can be minimized by spreading the daily carbohydrate quota over five or even six meals or snacks in the day. This will reduce the carbohydrate given at any one meal below 40 grams, and will ensure that the patient is never more than three hours without a carbohydrate-containing meal during his waking hours.

There is no objection to stabilizing the patient on a basal diet first, and then working up to the maintenance diet later, as recommended for cases treated with soluble insulin, except the question of time. It takes longer to stabilize a patient on zinc protamine insulin than on ordinary insulin, and for this reason it is suggested that the patient should be given a maintenance diet from the beginning. A specimen diet containing 2,000 Calories and 150 grams of carbohydrate, spread over six meals or snacks, is given in Diet No. 8 (p. 460). The statement made in dealing with the use of ordinary insulin, that it is easier to stabilize a patient on 150 grams of carbohydrate than on 200 grams, holds with even greater force for zinc protamine insulin.

As regards the initial dosage of zinc protamine insulin, we feel that it is a waste of time to begin with a small dose and increase this later. The initial dose for an adult should be 20 to 30 units depending on the severity of the case, and 15 to 20 units for a child. Since it takes two to four days for a dose, or any increase in the dose, to exert its full effect, alterations should not be made oftener than once in three days. As in the stabilization of a case with ordinary insulin, the examination of samples of urine passed at the definite times mentioned throughout the day will prove of great value in assessing the response to zinc protamine insulin and the necessity for altering the dose (see p. 245).

When the patient has been placed on the maintenance diet, 30 units of zinc protamine insulin are given as early after rising as is practicable. If, after three days of such treatment, all the urine samples are sugar-free, 30 units are obviously excessive and the dose should be reduced progressively by 4 to 6 units every three days until one or more of the urine samples show a trace of sugar. The dose reached at this point is the one required by the patient. At this stage blood-sugar tests carried out at intervals throughout the twenty-four hours are of value in confirming the stabilization.

If after three days glycosuria is still present, the dose of zinc protamine insulin should be increased by 4 to 6 units, depending n the degree of the persistent glycosuria. This procedure is repeated at three-day intervals until the pre-breakfast sample of rine becomes sugar-free, or until a maximum dose of about 50

units is reached. We do not consider it advisable for the gene
practitioner to attempt to control a patient's diabetes with o
single injection of zinc protamine insulin of more than this amou
If the patient's urine passed immediately before the mid-day m
still contains sugar with this dosage, the addition of soluble insu
to the dose of zinc protamine insulin is advised, as described belo
The absence of sugar in the pre-breakfast sample of urine indica
that the dose of zinc protamine insulin reached is sufficient
lower the blood sugar during the succeeding night to a normal le
or even lower. Hence no further increase in the dose must be ma
even if the other samples of urine contain sugar, otherwise hy
glycæmia will occur at night. If both the pre-breakfast sam
(No. 1) and the sample of urine passed last thing at night (No.
see p. 245) are sugar-free, it is advisable to transfer some of
carbohydrate from breakfast or the mid-day meal to the even
meal or the bedtime snack. If urine samples 1 and 4 still rem
sugar-free, the dose of zinc protamine insulin should be redu
cautiously.

In many cases of moderate severity, control of the diabetes
be secured by one daily injection of zinc protamine insulin.
severe cases, however, the dose of zinc protamine insulin gi
before breakfast may not control the glycosuria in the speci
passed between breakfast and lunch or even in the early aftern
although all subsequent specimens, including the pre-break
sample passed twenty-four hours later, may be sugar-free.
such cases most authorities favour giving both soluble insulin
zinc protamine insulin before breakfast, for this enables the
longed action of the zinc protamine insulin to be reinforced at
crucial times by the rapidly acting ordinary (soluble) insulin.

The patient is treated with zinc protamine insulin in increa
doses at three-day intervals until the maximal safe dose is fo
i.e. the pre-breakfast urine is sugar-free but some glycosuri
present at other times, particularly during the first half of the
An additional dose of ordinary insulin is now given along with
zinc protamine insulin. The initial dose of soluble insulin sh
be 4 to 8 units, this amount being increased progressively until
urine sample passed before the mid-day meal is sugar-free. Ex
in very severe cases, the urine will remain sugar-free throug
the rest of the day, as by mid-day the zinc protamine insulin
be coming into action. In extremely severe cases, a small do
soluble insulin may be necessary in the late afternoon as
According to Lawrence (1938) the simultaneous administratio

soluble insulin and zinc protamine insulin does not require two injections; both forms of insulin can be drawn up into the same syringe and injected together. The requisite dose of soluble insulin should always be drawn into the syringe first, so that the phial of soluble insulin (whose contents are acid) is not contaminated by any alkaline zinc protamine insulin from the needle. Other authorities do not agree that it is wise or desirable to mix the two types of insulin in the syringe, and recommend that they should be drawn from their phials and injected separately through a needle previously introduced through the skin. A point of practical importance is that if a mixture of the two insulins is being given to a patient whose pre-breakfast urine is sugar-free, the injection should be made immediately before breakfast and not shortly after rising, otherwise hypoglycæmia may result from the soluble insulin coming into action before sufficient carbohydrate has been absorbed from the food taken at breakfast.

Although the combination of soluble insulin and zinc protamine insulin as outlined above gives better control of a severe case of diabetes than does ordinary insulin alone, nevertheless it may be impossible to keep all samples of urine sugar-free without producing hypoglycæmia. In fact, in diabetes of great severity treated with large doses of zinc protamine insulin, a mild glycosuria last thing at night is desirable, as it indicates that hypoglycæmia during the night is unlikely to occur.

Before leaving the subject of zinc protamine insulin, it might be well to repeat the statement made already that this substance is apt to show an irregularity of action from day to day. A return of glycosuria in a patient who has been successfully controlled by zinc protamine insulin is not, therefore, an indication for an adjustment in insulin dosage unless the glycosuria persists for more than a day or two or is accompanied by ketonuria.

Treatment with Globin-Insulin

As has already been noted on page 243, globin-insulin generally produces its maximal effect on blood sugar from four to eight hours after injection. When hypoglycæmic reactions occur, it is usually within an hour of the mid-day meal. Accordingly, a buffer meal of 10 to 15 grams of carbohydrate should be prescribed about 11 o'clock, especially for persons doing heavy work. The quantity of carbohydrate taken at breakfast should not exceed 30 grams, for the globin-insulin does not begin to lower the blood sugar level materially for at least two hours after injection. The main carbohydrate meal should be in the middle of the day, as this is the time when the action of globin-insulin is at its maximum. As its action is passing off by bedtime, a buffer meal at this time, as recommended for zinc protamine insulin, is unnecessary. Diet Sheet No. 8, as given for zinc protamine insulin, will be found suitable for globin-insulin if 10 to 15 grams of the breakfast carbohydrate are taken at 11 o'clock, and the bedtime buffer meal of 15 grams of carbohydrates is taken at lunch time.

EXERCISE AND DIABETES

In a treated diabetic, muscular exercise causes a fall in the blood sugar. Therefore, the patient must keep in mind the possibility of hypoglycæmia if he has to indulge in any unwonted physica exertion, and he must be taught how to minimize the risk of thi complication. In cases receiving zinc protamine insulin it is wise for the patients to keep to the usual dose of insulin, but to tak an extra 15 to 20 grams of carbohydrate before or during th exercise, and another extra 15 to 20 grams in the evening, t prevent delayed onset of hypoglycæmia during the night followin the exercise. The carbohydrate may be taken as lump sugar (on lump is approximately 5 grams) or as any of the 20 to 25 gram carbohydrate feeds mentioned on p. 257. If the patient is bein treated with ordinary insulin, he may reduce somewhat the dos of insulin taken previous to the exertion or, alternatively, take 1 to 15 grams of carbohydrate as described above.

The inactivity imposed by the hospital regime even when th patient is not confined to bed may give a false idea of the patient insulin requirements on return to work. The need for readjustmen of diet and insulin must always be kept in mind after discharg from hospital to more active duties.

DIABETES AND PREGNANCY

Despite the acknowledged risks to both mother and fœtu pregnancy may be allowed when the parents wish to have childre provided skilled medical care is available. The risk is probably n justified on more than two occasions. The increasing demands f Calories, vitamins and minerals during the progress of pregnanc must be remembered. These may be met most easily by increasin the daily intake of milk in Diets 7 or 8 up to 1½ pints at the fourt month of pregnancy. The dose of insulin must be adjusted for th increased consumption of food, and it must be borne in mind that hypoglycæmia may occur towards full term as a result of the increasing production of insulin by the fœtus. Occasionally patients who need large doses of insulin during pregnancy need either none at all or much less subsequent to delivery.

INFECTION AND DIABETES

The presence of any infective disease or septic condition reduces the activity of insulin, whether endogenous or injected. Therefore, a diabetic patient developing any acute infection is in danger of developing ketosis and coma if prompt and suitable treatment is not given.

As was stated already, any diabetic patient developing an acute infection should go to bed and call in his doctor.

The dietetic treatment of a case of diabetes complicated by infection depends on whether or not the patient can take his ordinary diet. If he is too ill to do this, the light diet set out in Diet No. 9 (p. 462) should be given. This diet contains 150 grams of carbohydrate and 1,500 Calories. In a case of severe illness such as pneumonia, the patient will be unable to take even this diet and, instead, he should be given the diet recommended for the treatment of diabetic coma – 'the coma diet' – as described on p. 257.

As regards insulin dosage, it is clear that the patient's normal requirement will be increased during the infection. If the patient has been treated with ordinary insulin, injections should be given three or even four times daily, instead of twice. No hard-and-fast rules can be laid down about the dose of insulin which should be given. It is probably best to regulate the dose according to the degree of glycosuria present in a sample of urine passed just before the injection; the recommended doses are 20 to 30 units for a red reduction with Benedict's reagent, 15 to 20 units for a yellow reduction, and 10 to 15 units for a green reduction.

The patient treated with zinc protamine insulin presents a somewhat more difficult problem. It is probably best to continue with the usual dose of zinc protamine insulin, but three injections of ordinary insulin should be given daily as well, giving half the doses mentioned in the preceding paragraph, according to the results of urine tests before the injections. It should be stressed that, when marked ketonuria is present, soluble insulin is strongly advised whether alone or in combination with zinc protamine insulin.

SURGICAL INTERVENTION IN DIABETES

An operation involving the use of a general anæsthetic exposes a diabetic patient to the risk of developing ketosis owing to the action of certain anæsthetics on the liver, the effect of prolonged starvation and possibly vomiting. Special care is therefore required both before and after operation.

1. Pre-operative Treatment

1. *Non-emergency Cases.* When the operation is one of convenience, the patient's diabetic condition should first be brought under control. Even in mild cases, a liberal carbohydrate allowanc (150 to 200 grams) should be given, covered by sufficient insulin t allow the carbohydrate to be stored and metabolized, and no merely excreted. In this way, the glycogen content of the live will be increased and the toxic effect of the anæsthetic on the live cells reduced. As regards the choice of anæsthetic, chlorofor should never be given to a known diabetic owing to its hepatotox tendency, and ether is best avoided. Where possible, local or spin anæsthesia should be used. Nitrous oxide and oxygen is th general anæsthetic of choice.

On the day of operation, 30 grams of carbohydrate are give in the form of orange juice (5 oz. of orange juice plus ½ oz. glucose, diluted with water or as a solution of glucose flavoure with orange juice) three hours before operation, along with a do of ordinary insulin varying from 10 to 30 units depending on th severity of the diabetic condition. When there is a tendency t sickness, orange juice is not always so well tolerated as glucose.

2. *Emergency Cases.* When an operation has to be undertak within a matter of hours, the patient should be given 30 to grams of glucose, well diluted with water, and 25 units of insul should be injected. This treatment is given as long before operation as possible, up to a maximum of three hours. In emergen operations on patients with severe diabetes and ketosis, particularly on those unable to swallow, it is essential to introdu saline and glucose continuously by the intravenous drip metho insulin being given every three hours according to the findings urine or blood-sugar tests. Only in this way can the serious dang of dehydration, acidosis and circulatory failure be combated.

2. Post-operative Treatment

The patient will be unable to eat for some hours after operation, and vomiting during this period may add to the risk of ketosis. A simple way of treating such cases is to use the coma diet (described on p. 257) with six-hourly injections of ordinary insulin; if vomiting is so severe as to make it impossible for the patient to take even this diet, 30 grams of glucose (intravenously) and 15 units of insulin (subcutaneously) must be given every four hours. Some authorities recommend that severe cases of diabetes should receive an intravenous injection of one pint of 6 per cent glucose in saline and 15 units of insulin before returning to the ward from the operating theatre.

When recovery from the effect of the anæsthetic is complete, the patient can be given the light diet, Diet No. 9 (p. 462), until he is well enough to resume his ordinary diet.

DIABETIC COMA

Although the treatment of this emergency is, strictly speaking, outside the realm of the dietetic aspect of diabetes, except as regards prophylaxis, any discussion of diabetic treatment would appear incomplete without at least a brief reference to the measures to be adopted when faced with a case of diabetic coma. Unconsciousness in a diabetic patient is most likely to be due to either diabetic or hypoglycæmic coma, but other non-diabetic causes for coma must be excluded, such as uræmia or a cerebral vascular accident. The differential diagnosis between diabetic coma and hypoglycæmic coma is summarized in the Table on page 254.

If the cause of unconsciousness is diabetic coma, treatment must be started immediately, as every hour's delay makes the patient's chances of recovery more slender. Treatment should be undertaken only in properly equipped institutions, and by physicians specially experienced in this serious medical emergency. The remarkably low mortality rate of under 2 per cent obtained in recent years in Joslin's famous clinic is attributed by Root (1945) mainly to the recognition of the importance of these factors. Treatment should include the following:

1. *Insulin.* Owing to its rapid action, soluble insulin is the type that must be used. The initial dose should be large – *i.e.* 50 to 100 units – and in severe cases this should be repeated in two hours. Thereafter, smaller doses – *i.e.* 20 to 40 units should be

Symptoms and Signs	Diabetic Coma	Hypoglycæmic
1. Onset . . .	Gradual, with prodromal symptoms such as thirst, anorexia, abdominal pain, and vomiting.	Sudden, with coma in a fe utes. Then be convulsi maniacal ment befo coma deepen
2. Skin	Dry and cyanotic, with evidence of dehydration.	Normal colour ing common
3. Tongue . . .	Dry and furred.	Moist and cle
4. Temperature .	Usually subnormal at first.	Normal.
5. Respiration .	Deep and sighing ('air hunger'), with smell of acetone in the breath.	Quiet. No acetone breath.
6. Signs of dehydration . .	Marked – facies sunken, ocular tension reduced.	No evidence hydration.
7. Nervous system	Pupils usually normal but may be contracted. Reflexes absent in deep coma.	Pupils often Reflexes b there is bilateral plantar res
8. Urine . . .	Sugar + + + Ketone bodies + +	Sugar usuall although amount ma sent from ing in blad onset of co
9. Blood . . .	Blood sugar very high. Alkali reserve low.	Blood sugar kali reserv

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* If previous treatment has been with zinc protamine in onset of hypoglycæmic coma may be delayed and gradua convulsions (see p. 242).

given every two hours until the patient has regained cons the amount of insulin at each injection being regulat results of tests for glucose and ketone bodies applied t of urine obtained by catheterisation before the injec p. 251).

In the initial stage of treatment of severe cases, es of the blood sugar are of the greatest value in assess dosage.

Circulatory failure accompanies coma, with resul absorption of any substance injected subcutaneously.

vere cases of coma, half the first dose of insulin should be given avenously.

he importance of giving sufficient insulin at the outset of treat- t is clearly indicated by an analysis of cases recently treated losin's Clinic. The mortality rate for the last 123 cases of etic coma was 1·6 per cent, and the average amount of insulin n during the first three hours of treatment was 216 units; reas in his previous series of 525 cases, the mortality rate was er cent, and the average dose of insulin was only 83 units.

Glucose. The British practice in the treatment of diabetic has been to give glucose by mouth or intravenously. Usually am of glucose has been given for each unit of insulin. theoretical basis of this practice may be summarized as ws:

) It minimizes the risk of producing hypoglycæmia through overdosage of insulin.

It provides a large amount of glucose to be oxidized under the influence of the injected insulin, so that oxidation of the ketone bodies can proceed rapidly.

vertheless, if patients are treated under suitable institutional tions there appears to be no sound reason for this procedure; t the administration of glucose may actually be harmful. e glucose is already present in the patient's tissues, and onal glucose has been shown not to increase carbohydrate stion, but merely to make the assessment of the effect of d insulin more difficult. There is also some experimental ce to suggest that the intravenous infusion of large amounts r into patients already saturated with this substance may hepatic damage and anuria.

Fluids. The dehydration of diabetic coma constitutes almost a danger to life as the ketosis. It is one of the causes of the atory failure which is always present in diabetic coma, and, r with the loss of salts from the body which occurs in the preceding the coma, it leads to renal insufficiency.

dration must be treated energetically if the patient is to be rom complete circulatory failure and death. If conscious, ent may take fluid by mouth, in amounts of about 10 oz. ter hourly for the first twelve hours, or, preferably, half- th physiological saline if this does not cause vomiting. When tient is unconscious, or when vomiting is at all frequent, tial intravenous infusion of 2 pints of normal or double- th saline is strongly recommended. Owing to the circulatory

collapse, it will probably be necessary to cut down
and tie in a cannula. Subsequent administration of sal
by intravenous infusion, or by subcutaneous or rectal
amount of fluid given intravenously should not exc
(about 3·5 litres) in twenty-four hours.

The use of sodium bicarbonate solution to combat
is recommended by many authorities, and it is claim
especial benefit for those patients in whom there is mar
without severe hyperglycaemia. The sodium bicarbona
added to the intravenous glucose saline after this has
the suggested amount being ½ oz. (15 grams) to the
authors' opinion treatment with alkalis plays an insig
in the management of diabetic coma.

4. *Gastric Lavage.* In unconscious patients the emp
stomach through a soft rubber tube introduced via t
the greatest value; thereafter the stomach should be
and some normal saline left *in situ*. This should be
several occasions as long as gastric atony, dilatation a
can be demonstrated.

5. *General Measures to Combat Shock.* The circulat
diabetic coma must be treated on the same general
other form of secondary shock. The patient must be
bed, an electric cage being used if one is available, an
the bed may be raised. In severe circulatory collaps
of the vasomotor and respiratory centres in the medu
2 c.c., or methedrine 1 to 2 c.c., given intramuscularly
ously) may be of value. Since the collapse is essen
peripheral circulatory failure resulting from loss
electrolytes, the intravenous administration of gluco
be preferred. If this does not correct the circulat
transfusion of blood or, preferably, plasma should be
object of restoring the blood volume.

Once the patient is fully conscious and the immed
life is over, he should be given a coma diet – three
containing 20 to 25 grams of carbohydrate, with a
ordinary insulin six-hourly, that is before every sec
dose of insulin should be regulated by the results o
test on urine passed before each injection – 30 units
for a red reduction of Benedict's reagent, 20 units
reduction, and 10 units for a green reduction. Th
selected from the following, each of which contains
of carbohydrate:

angeade: Orange juice 3½ oz. (100 c.c.). Glucose ½ oz. (15
grams).
Water flavoured with lemon juice.
25 grams carbohydrate.
n porridge from ¾ oz. dry cereal (oatflour, cream of wheat,
barley flour).
t milk 5 oz.
5 grams carbohydrate.
ead ¾ oz.
k 5 oz.
ar 5 grams (1 teaspoonful).
grams carbohydrate.
ger's food ½ oz. (dry weight).
k 5 oz.
ar 5 grams (1 teaspoonful).
grams carbohydrate.
ltine ½ oz.
k 5 oz.
ar 5 grams (1 teaspoonful).
grams carbohydrate.

reatment is maintained until ketosis has been abolished for
twenty-four hours. After this, the patient is given the
etic diet shown on p. 462, with two or three injections of
insulin daily, and, finally, when appetite and strength
regained, his former diabetic diet and insulin dosage can
ed.

Hypoglycæmic Coma

tment of hypoglycæmia in its initial stages before un-
ness has developed is extremely simple, and consists of
inistration of carbohydrate by mouth in the amounts
on pp. 238 and 243. If the patient is in hypoglycæmic
en first seen by the doctor, an injection of ½ to
1000 adrenaline hydrochloride solution should be given
ously. The adrenaline will mobilize the glycogen stores
r, and thereby raise the blood sugar to a level which may
e patient to regain consciousness. He should then be
grams of glucose in solution as soon as he is able
r.

e patient is in deep coma it is unlikely that adrenaline by
produce recovery, in which case the adrenaline treatment

must be supplemented, as soon as the necessary appa assembled, by the administration of glucose solution g a Ryle's tube passed into the stomach through the n intravenous route. Whichever method is adopted, glucose should be given.

The danger of hypoglycæmia occurring insidious treated with zinc protamine insulin has already been p. 242. Such hypoglycæmia may be related causa creased frequency of diabetic coma noted by some o the introduction of this product. Its continued action the peripheral utilisation of glucose throughout th there is no compensatory intake of carbohydrate t glycogen stores in the liver, may lead to depletion o It is now believed that ketosis results from excessive fat, mainly in the liver, which occurs when live depleted.

DIABETES IN CHILDRE

It is fortunate that diabetes is less common in ch adults, for the treatment of diabetic children is und difficult than that of adults, for several reasons:

1. As the diet must be increased progressively growth, stabilisation with insulin has to be repeat the diet is changed.

2. The diabetic condition in a child seems to be l to an adult, and the carbohydrate tolerance varies fr Children also seem to be more sensitive to insulin that the tendency to marked variations in the bl of treated cases is greater.

3. The great susceptibility to infectious disease adds to the difficulty of treatment.

Diabetes in children always belongs to the mod grade, and no attempt must be made to treat a di diet alone, since the reduction of Calories and carbo be below the level required for normal growth and be constantly exposed to the danger of severe ket

The principles of dietetic treatment are the same with certain modifications.

Calorie Intake. This must meet the requirements allow for growth. The suggested Calorie intake for is given in the Table on p. 291. In the case of the d ge of 11, the lower figure in the range of Calorie values e taken, and even this figure may sometimes be reduced to 200 Calories without adversely affecting the rate of For adolescent diabetic girls and boys of 13 to 17 a Calorie f 2,300 to 2,500 and 2,500 to 3,000 respectively will be be adequate in most cases. Size, energy requirements and ctors must be taken into consideration in assessing the al needs of each individual case of diabetes. Additional ie portions of fat and protein (see Table 21) can be given d.

. Since protein is needed to form new tissue during there must be less protein restriction than in the case of It is probably best to give the normal protein quota for namely 1·4 grams per pound of body weight at the age of asing to 1 gram per pound of body weight at seventeen. drate. The allowance of carbohydrate should be 150 to s daily. The relatively greater intake in children than in necessary to combat the tendency to acidosis which is so n diabetic children.

stribution of carbohydrate throughout the day depends r the child is being treated with ordinary insulin or one nger acting insulins. It is best to avoid such foods as d jam, as the child may thus lose much of his natural r sugary foods.

de the requisite minerals for skeletal growth, the child given at least 1 pint of milk daily up to the age of nine, after 1½ pints.

treatment in children also follows the same principles as but the doses should be increased by smaller amounts owing to the child's greater sensitivity to insulin. There contra-indication to the use of zinc protamine insulin or ulin in children – in fact, one or other should be used possible, as many children require at least three in-f ordinary insulin daily to secure full control of their condition.

ar physical activity of children makes them prone to emia, and it is safer to adjust the insulin dosage so that always contains a trace of sugar.

nt of a diabetic child can only be considered successful tion to the control of the diabetes, a satisfactory gain in ght and mental development occurs.

WAR-TIME DIABETIC ALLOWANCES

The diabetic's difficulty in metabolizing carbohydrate food h
been recognized by the Ministry of Food and special allowanc
have been made of other foods on the advice of the Food Rationi
(Special Diets) Advisory Committee of the Medical Resear
Council. Thus, the protein allowance has been increased by t
extra rations (3s. 6d. worth of meat weekly), the fat allowances
two extra rations of butter or margarine (18 oz. weekly), while t
cheese ration is 12 oz. weekly, which is four times the ordin
cheese ration at the time of writing. In addition, a priority rat
of 1 pint of milk daily is given. No extra rations of bacon or e
are granted and this necessitates careful thought on the provis
of exchanges for these foods which are so useful in construct
the diabetic's breakfast or supper. Table 25 (p. 446)
supply certain exchanges when neither bacon nor eggs (shell
dried) are available. Every diabetic should be instructed to use
whole of his priority ration of cheese. As a substitute in war-ti
for the orange, apple, pear or ½-grapefruit shown on the I
sheets, ½ to ¾ oz. of bread or 1½ oz. of potato should be used.
loss of vitamin C resulting therefrom will be of little conseque
provided that the patient is taking ample green vegetables at
of his meals. Should there be any reason to suspect a deficienc
vitamin C in the diet, one tablet containing 25 milligrams of asco
acid may be given daily.

CHAPTER XII

DISEASES OF THE ALIMENTARY TRACT

INTRODUCTION: GENERAL CONSIDERATIONS

DISEASES of the alimentary tract may be organic or functional. While textbooks of medicine devote most of their space to organic disease because of its serious significance, it has been generally believed in the past that dyspepsia of functional origin occurs more frequently. The widespread use of the gastroscope in recent times suggests that many cases of dyspepsia which would formerly have been called functional, because alterations in the secretory or motor activity of the stomach were present but no structural changes could be demonstrated by radiological examination, should in reality be classified as organic because of the visible evidence of gastritis. There is good reason to believe that organic lesions may develop if abnormal functional states continue over a long period. For instance, the sequence of events in peptic ulcer would frequently appear to be hyperchlorhydric dyspepsia caused, for instance, by overwork and worry, which passes on first into a hypertrophic erosive gastritis and eventually into ulcer formation. In our experience, unless gastric rest is obtained by correcting the general physical and psychological strain, the ulcer will tend to relapse, no matter what dietary restrictions are ordered. This conception is of great importance because it entails the acceptance of the view that the restoration of the physiological working of the alimentary tract by the correction of faulty habits and anxiety states is an essential part of the prevention and treatment of both organic and functional disease, and is as important as the diet or drugs prescribed.

Accordingly it must be obvious that every patient suffering from a disease or disorder of the alimentary tract must receive the most careful questioning in regard to his dietary history and social habits. This includes information about the number and time of the meals taken daily, whether they are taken regularly, if they are hurried, and if the food is adequately chewed. A description must be obtained of the various foods eaten and of their effects in alleviating or producing the symptoms. Particular inquiry must

be made into the taking of excessive quantities of alcoh
especially on an empty stomach, strong tea or coffee at t
frequent intervals, or indigestible articles of food and condimen
(see p. 265). A careful history should be obtained with the obje
of finding out whether the patient is living in a state of tensi
consequent on anxiety induced by abnormal social relations
home or at work. We have repeatedly seen dyspepsia developi
or ulceration recurring as a result of unhappy marital relatio
where a love affair is not proceeding on a satisfactory course, wh
an employee is being bullied by an employer, or where so
financial misfortune is causing undue worry.

Lastly, in diseases of the gastro-intestinal tract, common se
dictates that a careful investigation should be made of the tee
with the object of determining, first, if they are mechanica
efficient and, secondly, if the stomach is being poisoned eit
locally by direct infection or generally by systemic intoxicati
The reduction of foodstuffs to a soft pulp by efficient masticat
is a fundamental prerequisite to good digestion. It is essent
therefore, to determine whether the patient has an adequ
number of teeth in his mouth, and whether those which
present are directly in apposition in the lower and upper jaw.
dentures are used, they must be comfortable, otherwise they ma
utilized for æsthetic purposes at social functions, but removed
mealtimes.

If dental sepsis is present, it must be treated. This is a prob
of great difficulty and one which must be resolved according to
circumstances in each individual case. In the presence of ac
organic disease of the alimentary tract, it is probably wiser t
content with conservative methods until the patient's gen
condition has improved, when extraction of teeth can be car
out if it should be considered desirable. The same general princi
apply to nasopharyngeal sepsis.

When prescribing a diet for a patient suffering from any dis
of the alimentary or indeed any other system, it is essentia
bear in mind the following points. (1) Be sure that the diet is a
balanced one, and is not only satisfactory in relation to Calo
fat, protein and carbohydrates, but is also adequate in vita
and minerals. If necessary, vitamin concentrates and minerals
be added. (2) Remember, if attention is directed solely to
dietetic treatment of a particular symptom – *e.g.* diarrhœa, ga
pain – the general nutrition of the patient, and with this his ab
to resist infection and repair ulceration, may suffer. (3) Cons

the patient's occupation and make arrangements that the dietary regime ordered is suitable in each individual case. Obviously different schemes will be required for an office worker, a miner, and a night watchman. (4) Give due consideration to the individual's likes and dislikes for different articles of food. Never prohibit an article from a diet unless there is a clear history of a personal idiosyncrasy to it, or unless there is adequate clinical or experimental evidence for its exclusion. (5) If the diet advised is materially different from that which the patient is accustomed to take, the change should, if possible, be made gradually and not abruptly. This is particularly the case in diabetes, or in constipation if large amounts of roughage in the form of fruit or green vegetables are ordered. (6) Remember that the dietetic requirements of acute and chronic disease may be very different. Thus, in intense diarrhœa, excessive vomiting or infections which are severe in degree but short in duration, *e.g.* lobar pneumonia, the essential need is for fluids, salt and glucose to prevent or correct dehydration, alkalosis or hypochloræmia, toxæmia and ketosis. In long continued disease, such as pulmonary tuberculosis, typhoid fever or ulcerative colitis, the diet must be adequate both quantitatively in Calories and qualitatively in the various essential food constituents.

When the family doctor is consulted for dyspepsia he should make a careful inquiry into the dietary history and social habits, as discussed in the opening paragraphs of this chapter. Thereafter he should make a general physical examination, including the abdomen. Unless he finds reasons for believing that the dyspeptic symptoms indicate some serious organic disease, such as cancer of the stomach, it is unnecessary and probably unwise initially to investigate the stomach by test meal or X-rays. In most cases it will be found that the patient has been overworking or overworrying, or is bolting his meals or eating his meals when excessively tired, or has been smoking immoderately or taking too much alcohol. The patient should be assured that if he carries out the doctor's advice and takes a bland diet for a few days the dyspepsia will probably clear up rapidly. The summary of instructions given below may be of help to the doctor in reminding him of the various points of importance. The specimen bland diet (p. 266) made up from the foods permitted should be prescribed. If the dyspepsia ails to clear up or a relapse occurs within a short time, despite dherence to instructions, it is advisable to place the patient in n institution where a full investigation of the alimentary tract can be undertaken. If, on the other hand, the dyspepsia entirely c appears, it is most desirable that the restrictions on foods wh were initially imposed should be gradually relaxed. The pati with functional dyspepsia, gastritis or peptic ulcer who has b correctly treated should be able to eat almost any article food, provided that he has learnt to chew thoroughly and ta plenty of time over his meals. It is unnecessary and unwise such patients to smoke excessively, to take alcohol on an emp stomach, or to drink very strong tea or coffee. There is no r hardship in giving up pickles and taking condiments sparing On the other hand, it *is* a real hardship both to the patient and wife if he is permanently prohibited from taking soups or fried fatty dishes. Again, such common articles as sausage, bacon a twice-cooked meats fill an important place in the diets of worki class families. If eaten slowly, masticated well and taken moderate quantities, they are well tolerated. Similarly, r vegetables such as tender lettuce leaves and ripe tomatoes, a cooked coarse vegetables, should not be prohibited permanen Common-sense avoidance of the coarse fibrous veins and the of the sieve can make these valuable foods suitable for the majo of patients who carry out the doctor's instructions.

Summary of Instructions for a Patient with Dyspeps

1. Take four meals a day.
2. Take your meals at regular times daily.
3. Do not hurry your meals; chew your food carefully.
4. Avoid rush and hurry before and after meals. If poss rest for a few minutes before and after eating.
5. See that you get sufficient sleep at night.
6. Remember that anxiety and worry can upset digestion.
7. Avoid large, heavy meals and any article of food which find disagrees with you.
8. Avoid foods which are mechanically irritating or chemic stimulating (see list below), and very hot or very c foods.
9. Do not smoke or drink alcohol before meals, when stomach is empty.
10. Limit the fluid drunk with meals, but drink plenty of w between meals.
11. Attend to your bowels (see p. 287).
12. Consult your dentist at regular intervals.

Avoid the following foods which experience has shown are either intrinsically indigestible, mechanically irritating or chemically stimulating.

1. Alcohol, aerated mineral waters, strong tea and coffee, gravies, soups, particularly those rich in meat extracts.
2. Raw vegetables, salad, celery, cucumber, water-cress, tomatoes, mushrooms.
3. Raw unripe fruit, and dried fruit (*e.g.* currants, raisins and figs), nuts, and the pips, skins and peels of all fruits, whether cooked or in puddings, cakes or jam.
4. Pickles, spices and condiments.
5. Tough, twice-cooked, or highly-seasoned meats, including sausages, bacon and pork.
6. Smoked and fried fish, and fatty fish such as herring, mackerel, salmon and sardines.
7. Rich and heavy puddings, and fried foods in general.
8. New bread and scones, hot buttered toast, wholemeal bread or biscuits, rye or wheat crisp bread, coarse cereals, pastry, cakes containing dried fruit or peel.

The following foods are permitted:

1. Weak tea at two meals only.
2. Dairy products, *i.e.* milk, cream, butter, cream cheese, eggs (not fried).
3. Fish – white fish, steamed or boiled.
4. Meat – sweetbreads, brains, tripe, chicken, rabbit, tender underdone beef or lamb.
5. Crisp toast (buttered cold), rusks and stale white bread.
6. Plain biscuits and cakes, or sponge cake. Honey, golden syrup, jellies.
7. Cereals – refined and well cooked, *e.g.* cornflour, semolina, ground rice, and oatflour porridge.
8. Puddings – junket, jellies, custards, blancmange.
9. Vegetables – potatoes, boiled and baked, and green and yellow vegetables finely sieved and puréed with butter.
10. Fruits – finely sieved and stewed, and served as purées or fools, and fruit juices strained, sweetened, and diluted with water, or used in jellies.

Specimen Bland Diet

Breakfast	Refined cooked cereal, small helping (if desired). 1 or 2 eggs, lightly boiled, poached or scrambled. Crisp toast or rusks, and butter (or vitaminized margarine) and jelly. Weak, milky tea.
Lunch	Boiled or steamed white fish, or sweetbreads or tripe. Mashed potatoes, puréed vegetable. Milk pudding.
Tea	Toast, biscuits or rusks. Butter (or vitaminized margarine), honey, or jelly. Sponge cake. Milky tea.
Dinner	Milk soup or strained vegetable soup (small helping). Breast of chicken, rabbit, or underdone beef or lamb (small helping). Baked potato, puréed vegetables. Stewed apples or fruit jelly, with milk or cream.

Before bed a glass of warm milk, Ovaltine or Benger's food.

Fruit juice with water should be taken with or between meals.

PEPTIC ULCER

Before discussing the medical and dietetic treatment of peptic ulcer, it is necessary to indicate the types of case for which a surgeon should always be called into consultation. These types are:

1. The old, indurated ulcer of the duodenum which is producing pyloric stenosis and marked gastric retention, and the gastric ulcer on the lesser curvature producing such a degree of 'hour-glass' contraction of the stomach as to lead to obstructive symptoms.

2. (*a*) Any gastric ulcer in middle-aged patients which does not show satisfactory signs of healing within 4 weeks while on strict medical treatment. (*b*) Ulcers of the pyloric antrum and pyloric canal, because of their known tendency to become malignant. (*c*) Ulcers of the greater curvature which are almost invariably malignant from the beginning.

3. Ulcers which show a persistent tendency to bleed, in spite of repeated courses of adequate medical treatment, particularly if the patient is over forty years of age and suffers from arteriosclerosis.

Principles of Treatment

The essentials are: (i) Rest (physical). (ii) Relaxation (psychological). (iii) A bland diet given in small amounts at frequent intervals.

Antacids and atropine are of subsidiary importance but are of great value for the control of the symptoms of dyspepsia and pylorospasm when these are present. Such symptoms usually disappear within two or three days of the commencement of treatment provided no complications are present, *e.g.* pyloric stenosis or adhesions to the pancreas or gall-bladder.

The Acid Factor is of undoubted importance in the ætiology of peptic ulcer, since ulceration only occurs when hydrochloric acid is present in the stomach. Peptic ulcer only develops at sites (gastric and extragastric) where acid comes in contact with mucous membrane. In addition, the frequency with which peptic ulcer and hyperacidity co-exist is highly suggestive. It is also widely held that hydrochloric acid is the most important factor in the prevention of healing of ulcers, and the chief object of nearly all recognized types of treatment is the control of the acid factor. We believe, however, that this aspect has been somewhat exaggerated, because it has long been recognized that at the end of a course of treatment, when the ulcer has healed, the acid content of the stomach may be greater than when treatment started. Also, many ulcers heal when a reorganization of the patient's manner of living is effected, without treatment being given for the control of gastric secretion. Lastly, Palmer has shown that ulcers will heal if the patient is confined to bed on a bland diet and given, in addition, various drugs and foods which are known to be stimulants of gastric secretion.

At the present time, however, it is probably wise to accept the view that hyperacidity tends to delay healing, and accordingly to utilize all the means at our disposal for reducing the secretory activity of the stomach and neutralizing, as far as possible, any excess of acid.

Motor Factor. Little is known about the effects of hypermotility in the causation and prevention of healing of peptic ulcer. We feel that the motor factor may be of more importance than the acid factor. Excessive and irregular peristalsis and pylorospasm may produce ischæmia in, and thus devitalization of, the mucous membrane which is accordingly more susceptible to the action of hydrochloric acid and pepsin. Further, the exaggerated muscular movements tend to cause mechanical damage to the lining of the stomach, particularly on the 'magenstrasse', through the forcible impact of the gastric contents. Lastly, the motor factor is of importance when an ulcer has developed, since local rest for the attainment of healing is as important in the stomach as in any other situation in the body.

For the attainment of control of secretory and motor activity of the stomach the two chief essentials are mental and physical rest, and the use of a bland diet given in small amounts at frequent intervals, the constituents of which are mechanically non-irritating and chemically non-stimulating.

Rest, Local and General. It is widely held that rest in bed for a period of four weeks is an essential part of the medical treatment of every case of peptic ulcer. By this means alone many ulcers will heal satisfactorily on an ordinary ward diet.

Rest in bed is of the greatest value in raising the powers of resistance of a patient who is suffering from physical exhaustion consequent on overwork in the office, factory or home. Rest produces its beneficial effects not only on the body as a whole but also locally on the stomach. It is generally advisable that this period of rest in bed should be undertaken in a hospital or nursing home, and not in the patient's house. The ulcer regime is carried out more effectively in these circumstances, and the patient accepts more quickly and more easily the discipline and the inconveniences which he must undergo for a period of weeks when he may be feeling perfectly well. Lastly, he is removed from many petty causes of domestic worry and irritation, which keep up a state of nervous tension and must accordingly be controlled if satisfactory healing is to be obtained. Under such circumstances sedative drugs, such as phenobarbitone (½ gr. twice or thrice daily), are, in our experience, often of more value than alkalis and atropine. During the period of institutional treatment the physician has ample opportunities for investigating the psychological factors of ætiological importance and of making the adjustments in the patient's social relations which will be necessary on his return to his home and work. At the same time education can be given upon the physiological methods of eating, the types of food and drink to be taken and avoided, the care of the teeth, etc., as outlined on p. 264.

The advantages of rest in bed are obvious, but we are not able to agree with the statement, frequently made in textbooks, tha rest in bed is invariably essential or, in certain circumstances, even the best line of treatment. For instance, if a married woman with three young children is admitted to hospital for treatment and is continuously worrying about whether the children are being properly looked after at home, better results may be obtained by treating the patient in her own house. In which case arrangements must be made for relieving her of some of her household duties, and thus enabling her to get modified rest. Again, if a patient with an ulcer is in fear of losing his employment because of being off duty for a period of one or two months, the anxiety state resulting may more than counterbalance the benefits of rest in bed, in which case ambulant treatment with modified rest, arranged according to the individual circumstances, should first be tried as it may be very effective. At the present time there are probably more cases of duodenal ulcer treated in out-patient departments by ambulatory methods than are treated as in-patients in the wards, and the majority heal satisfactorily as judged by relief of symptoms and radiological examination. In view of these facts it is unwise to claim that rest in bed is essential, although it is probably desirable in the majority of cases. We feel that, if ambulatory treatment is advised, it should be limited to cases of duodenal ulcer, since gastric ulcers, owing to their tendency to bleed or undergo malignant changes, require the most careful and repeated observation.

Diet in Peptic Ulcer

During the past thirty years innumerable schemes of diet have been advocated for the treatment of peptic ulcer. In all types the diet is bland and given in small amounts at short intervals. Since the percentage of good results claimed for each type of diet is approximately the same, it would appear that these two essentials are of more importance than the actual food constituents, which differ widely in the various schemes advocated. The diets used at the present time can be classified into three main types. 1. The Sippy diet and its modification by Hurst, which consists essentially of hourly feeds of small quantities (5 oz.) of milk, cream or other milky food. 2. The Lenhartz diet, which is also given mainly in fluid form but contains more protein than the Sippy diet supplied in the form of eggs switched with milk. 3. The Meulengracht diet, which includes a much wider variety of foodstuffs, *e.g.* chicken, rabbit and other meats, fruits and vegetables, all the food being carefully pounded or sieved. The feeds are given at two-hourly intervals.

It is claimed by the advocates of the Sippy type of diet that complete neutralization of the hydrochloric acid in the gastric contents throughout the waking hours is obtained if, in addition to the hourly feeds of milk or milk pudding, antacids after and atropine and olive oil before a number of the meals are given. Nicol, working under one of us, has shown that this is not the case. In twenty consecutive patients suffering from peptic ulcer and hyperchlorhydria placed on this regime, from whom samples of gastric juice were removed at hourly intervals throughout the twenty-four hours, free hydrochloric acid was frequently present. The mean acidity obtained by averaging the figures for free hydrochloric acid in the twenty-four samples was actually higher in every case on this regime than when the patient was on two-hourly feeds of the Meulengracht type or on four light meals a day. For this reason, and because of the inconvenience of hourly feeds to both the patient and nurses, we are satisfied that the time intervals between feeds should be two hours and not one.

The chief differences between the Meulengracht and the Sippy types of diet is that the protein content is considerably higher in the former. Contrary to the general belief that proteins should be restricted in ulcer diets because of their tendency to stimulate the secretion of hydrochloric acid, we have found that the giving of protein foods, such as pounded fish, chicken or eggs, reduces the hydrochloric acid much more efficiently and maintains it at a low level for longer periods than do meals consisting of milk and cream. The Meulengracht type of diet is more acceptable to the patient because it contains, from the beginning of treatment, a much wider variety of foods than either the Lenhartz or the Sippy. Many of the dishes allowed are in a semi-solid puréed form, and this we have found to be of importance because they remain longer in the stomach than fluids, and hence are much more effective in neutralizing and binding hydrochloric acid.

In his most recent paper, Meulengracht claims, from his experience of 491 cases of peptic ulceration accompanied by severe hæmorrhage, that the lesions healed more rapidly and more effectively when treated by his methods than when the more restricted type of Sippy diet was used, and that the mortality from hæmatemesis had fallen from 9 per cent to less than 2 per cent since the adoption of a fuller diet. If such a diet can be given to acutely ill patients with active ulceration and bleeding, it must be equally suitable, and much more acceptable, to patients who have less severe ulceration and are not suffering from shock. For several years now we have treated patients suffering from peptic ulcer with and without hæmatemesis, from the beginning with a modification of the type of diet recommended by Meulengracht, and we can confidently recommend its use, both because of the excellent results obtained and because of the satisfaction given to patients and nursing staff.

First Stage. The speed with which peptic ulcers heal varies greatly from case to case, but it would be unwise to assume that, in general, a firm scar can be obtained in less than two months. We do not approve of 'step-ladder' diets in which the amount of food is increased automatically at daily or weekly intervals. Such a method assumes that healing occurs at the same speed in every case. Accordingly we advise a bland diet, well balanced and varied in quality, which should be taken two-hourly without alteration for at least one month. The basis of the diet is that adequate amounts of protein in the form of eggs, tripe, sweetbread, fish, chicken and rabbit, should be taken as purées, soufflés and creams, at three or four meals in the day, and milk and cream or a milky food should be given midway between these four meals (see Diet No. 10, p. 463).

If symptoms of dyspepsia or pylorospasm are present, atropine 1/100 gr. in water should be given before the main meals, and last thing at night, and ½ to 1 teaspoonful of an antacid or alkaline powder ½ to 1 hour after the four main meals and last thing at night. For the control of muscular spasm atropine must be pushed to the limit of tolerance as indicated by a dry tongue and paralysis of accommodation. In patients on ambulatory treatment, the disadvantages and dangers of atropine must be recognized, particularly if the patient is a motor-driver. Our investigation by the method of 24-hour test-meals has shown that atropine, even in highly toxic doses, has an insignificant effect in the control of acidity. Accordingly its use can be discontinued when the symptoms of painful contraction and spasm have disappeared.

Many authorities, including Meakins, Kantor and others, believe that, when symptoms have disappeared after rest in bed, alkalis or antacids can be dispensed with. As has already been indicated above, our own experience with the 24-hour test-meal shows that alkalis are of doubtful value in the control of hyperacidity. In view, however, of the divergence of opinion on this subject at the present time, the practitioner is at liberty to use antacids or not as he thinks fit, provided that he realizes that the fundamentals of treatment are diet, mental relaxation and physical rest. Attention is drawn to the obvious advantages of the neutral antacids, magnesium trisilicate and aluminium hydroxide, which are as effective as the alkalis usually employed, *e.g.* sodium bicarbonate, magnesium oxide, and calcium carbonate, in neutralizing hydrochloric acid without producing a secondary secretion of hydrochloric acid or inducing alkalosis. On the other hand, for the immediate symptomatic relief of hyperchlorhydric dyspepsia, baking soda, chalk and magnesium oxide are probably more effective than the above-mentioned antacids, and considerably cheaper.

Intermediate Stage. The diet recommended for the second month consists of the same articles of food given at the same intervals, but the quantities should be increased. Fish, chicken and rabbit can be given boiled or steamed instead of in the form of purées, and small helpings of tender beef or mutton are allowed.

The *Post-ulcer Regime* should be continued for at least two years, and, better still, for life. It consists of an ordinary diet with the exclusion of certain articles of food which are particularly irritating or stimulating (see p. 265). The most essential feature of the post-ulcer regime is the instruction of the patient in physiological methods of eating and living (see pp. 261 to 265). He should never go longer than 2 to 2½ hours without taking some food during the daytime. This is accomplished by taking a glass of milk or milky food and a biscuit between breakfast and lunch, and between lunch and dinner if he is not accustomed to taking tea, and last thing at night. Drugs are unnecessary provided there are no symptoms.

HÆMATEMESIS

Until recently, the treatment of hæmatemesis consisted of absolut physical rest in bed, the injection of morphia, and the prohibitio of all food and fluid by mouth for 24 to 48 hours. Thereafter th patient was allowed to sip small amounts of water or milk, and th diet was gradually stepped up to the Sippy or Lenhartz die according to the physician's inclination. Exclusion of food an fluid by mouth was based on the erroneous view that gastric res was thus best obtained. Recent investigations have shown tha gastric peristalsis is at its minimum when the stomach contai food and not when it is empty. In 1931, Meulengracht came to th conclusion that the withholding of food and fluid from patien who were suffering from post-hæmorrhagic shock was contrary t physiological principles. Accordingly he adopted a method treatment which permitted the patient to eat a varied and liber bland diet *ad libitum* immediately after the hæmorrhage. He is satisfied that the good results obtained do not depend on any special diet but on the supplying of adequate Calories, water, salts, and vitamins in a bland form. There are two reasons for favouring the adoption of the Meulengracht regime. First, studies of the water and nitrogen intake and output in patients suffering from gastro-duodenal hæmorrhage, and who were being treated by starvation or semi-starvation, show that a negative water and nitrogen balance is present to a marked degree. Secondly, practical experience has proved that patients prefer the Meulengracht regime and take the diet well, that recovery is more rapid, that the bleeding is neither increased nor prolonged, that the mortality is reduced, and that the work of the medical and nursing staff is lightened and simplified. Our own experience of the value of giving fluid and bland food from the beginning of treatment is similar to that of Meulengracht, and accordingly we recommend that the first stage diet (see Diet No. 10, p. 463) advised for the treatment of uncomplicated peptic ulcer is equally suitable for patients suffering from hæmatemesis. The correction of the dehydration is of even greater importance than the correction of the negative nitrogen balance, for it has been shown that the old regime of restricted fluid intake was not uncommonly followed by uræmia and death, the blood volume being too low for the kidneys to maintain normal efficiency. The patient should therefore be encouraged to sip water or citrated milk as freely as possible. If significant dehydration and shock are present, additional fluid must be given per rectum or intravenously. In very ill patients with nausea and exhaustion who have no desire for food, it is unnecessary to force it on them; milk, milky foods and egg and milk flips in 5 oz. feeds at two-hourly intervals will be adequate for a day or two under these circumstances. In patients who have no disinclination to eat, the first-stage ulcer diet should be given from the start.

GASTRITIS (GASTRIC CATARRH)

The introduction in recent years of the flexible gastroscope, improvements in radiological technique and special methods of fixation of specimens for histological investigation have clearly demonstrated that gastritis occurs much more frequently than was formerly believed. Gastritis may result directly from toxins, bacterial (influenza, pneumonia, pyogenic infections, etc.) or metabolic (uræmia, diabetes, etc.), which reach the stomach via the blood stream (hæmatogenous gastritis) or by direct irritation of the mucous membrane of the stomach by ingestion of substances which are physical or chemical irritants (irritation gastritis), or indirectly by venous congestion secondary to cardiac failure or cirrhosis of the liver. The factors of particular importance in irritation gastritis are indigestible articles of food, alcohol, infected articles of food, drugs (*e.g.* salicylates, iodides, heavy metals, etc.), and more rarely foods to which the patient has a specific idiosyncrasy. Lastly, it should be mentioned that evidence has been accumulated in recent times to show that a slowly developing achlorhydric gastritis may result from prolonged dietary deficiency, particularly of proteins and vitamins A and B.

Gastritis may be acute or chronic. In the former the secretion of hydrochloric acid is invariably reduced, and in the latter it ma be increased or reduced. Superficial hypertrophic gastritis is frequent accompaniment of peptic, especially duodenal, ulcer. I the early stage of irritation gastritis, consequent upon the ingestio of irritating food or drink, the gastroscopic appearances are thos of congestion and œdema. If the irritation is extreme or long continued, and particularly if it acts on the stomach of a perso with the hyposthenic diathesis, achlorhydric gastritis eventuall results.

Treatment

1. *Acute Gastritis.* Acute gastritis is recognized in severe infectio diseases by the occurrence of nausea and vomiting, while acu irritation gastritis is more frequently a concomitant of food poiso ing or unwise indulgence in alcohol or rich and indigestible articl of food in large quantities.

The principles on which the treatment is based may be su marized as follows: (i) the neutralization or removal of the cau irritant by the appropriate means – antisera, gastric lavage, cast oil, specific antidotes, etc.; (ii) the prescription of sedative dru *e.g.* alkalis, bismuth and opium or one of its alkaloids, if ac intra-abdominal complications can be eliminated; (iii) the wit holding of all foods for 24 to 48 hours; and (iv) the prevent or correction of dehydration and hypochloræmia. Since ac gastritis with severe vomiting rarely lasts more than two or th days, attention need not be directed primarily to the ma tenance of the patient's nutrition as is essential in chr gastro-intestinal diseases, but should be focused on the prevent of the serious complications of dehydration and hypochlora which follow rapid fluid loss. While all food should be withheld, water or $\frac{1}{2}$-normal saline with or without the addition of glucose or fruit juice should be frequently given. Small amounts (3 to 6 oz.) may be taken every hour. If the patient is unable to retain such fluids given by mouth, and signs of dehydration appear, normal saline or 5 per cent glucose in normal saline must be given rectally or parenterally by the drip method. With improvement of the patient's condition, small feeds of barley water, glucose water, whey or glucose lemonade may be begun. With further improvement citrated or peptonized milk can be given, and thereafter the dietetic ladder can be gradually climbed by the addition of Benger's food, arrowroot, cornflour, custard, and fruit jellies, until first the dietary regime recommended under chronic gastritis and later a normal diet is reached.

2. *Chronic Gastritis.* No matter whether chronic gastritis is associated with hypo- or hyper-secretion of hydrochloric acid, the *immediate* underlying principle of treatment is the same, namely, rest to the chronically inflamed gastric mucous membrane. The application of this principle may be summarized as follows: (i) removal or correction of the underlying causative factor, whether toxic, infective, physical or nervous in origin; (ii) correction of faulty habits of eating (see pp. 261 to 265); (iii) the administration of drugs – sodium bicarbonate, bismuth and magnesium carbonate, ferrous sulphate if anæmia is present, and hydrochloric acid and pepsin when the inflammation has subsided if free hydrochloric acid has not returned; (iv) gastric lavage with 1 to 4 drachms of hydrogen peroxide (10 vols. per cent) to 1 pint of water, if free hydrochloric acid has not returned within a few weeks and excess of mucus is present; and (v) a bland diet given in small quantities at two-hourly intervals, mechanically non-irritating but adequate in Calories, minerals and vitamins. In chronic gastritis in which symptoms of pain and flatulence are marked, the first stage ulcer diet (see Diet No. 10, p. 463) is indicated, and it may be advisable to keep the patient in bed for one or two weeks. In the majority of cases, however, rest in bed is not required, and the intermediate stage diet (see p. 272) can be used provided the patient can be trusted to chew his food thoroughly and eat slowly.

If free hydrochloric acid has not returned in a few weeks or is present in reduced amount despite gastric lavage and the dietary regime recommended above, a stimulating diet should be recommended on the lines advised for functional achlorhydric dyspepsia (see Diet No. 11, p. 464). When the symptoms of gastritis have disappeared and the evidence of catarrh of the mucous membrane, namely excessive mucus in the gastric juice, is absent, the patient can return to an ordinary diet provided he obeys the general instructions as to physiological methods of eating given on pp. 261 to 265.

FUNCTIONAL DYSPEPSIA

1. Hyperchlorhydric Dyspepsia. Acid Dyspepsia

Functional dyspepsia may be defined as that state which produces abnormal sensations in the abdomen related to the ingestion of food, but is not accompanied by or due to any structural change in the stomach itself.

Hyperchlorhydria may be defined as a condition in which th concentration of free hydrochloric acid in the stomach is highe than normal. The excess secretion of acid may be dependent o a constitutional state or may be conditioned by emotional, irritative, toxic or reflex causes. It may initiate or follow organic diseas of the stomach or duodenum, *e.g.* peptic ulcer, duodenitis or antra gastritis. From this it follows that anatomical lesions may b absent or present. In addition, hyperchlorhydria may be symptomless or associated with symptoms identical with those present i peptic ulcer, but usually of less intensity. The term hyperchlor hydria should be restricted to an excess of hydrochloric acid in th stomach without symptoms, and acid dyspepsia or hyperchlor hydric dyspepsia used when symptoms are present.

In every case treatment must be preceded by a search for th cause of the hyperchlorhydria. This includes (i) the exclusion c local disease in the stomach and duodenum by appropriate means (ii) an investigation into the causes of anxiety states – overwor and worry, excessive fatigue, whether physical or mental; (iii) a inquiry into the patient's habits in regard to food, alcohol an smoking, as indicated on pp. 261 to 265; (iv) an examination of th appendix, gall-bladder, colon, etc., since it is well recognized tha hyperchlorhydric dyspepsia can be caused reflexly by disease i adjacent organs.

If no satisfactory explanation is forthcoming after investigatio have been made on the lines suggested above, it is justifiable t assume that the increased secretion of acid is due to an inborn err which is believed to occur in 10 per cent of persons, and which h been termed by Hurst 'the hypersthenic gastric diathesis'. Sin it has been proved that the healthy mucous membrane of the stomach is insensitive to the introduction of hydrochloric acid in a concentration as high as 0·5 per cent, it follows that, if symptoms of dyspepsia are present in the absence of damage to the mucous membrane, they are probably due to excessive and irregular peristalsis resulting from central nervous impulses consequent upon undue emotion or anxiety.

Treatment consists of (i) removal or correction of the causal factors; (ii) the correction of faulty habits of eating and drinking (see pp. 261 to 265); (iii) the administration of drugs – sedative, antispasmodic and antacid; (iv) psychological treatment for the correction of faulty methods of living at home and at work, and the removal of causes of anxiety and tension; and (v) the provision of a suitable diet which should be bland in character, adequate in Calories, minerals and vitamins, mechanically non-irritating and chemically non-stimulating, and given in moderate amounts at relatively short intervals. Psychotherapy includes the mental effects produced on the patient by taking a careful history of the digestive troubles and of family worries, and the making of a complete physical examination; to quote Alvarez – 'From the mental purgation that results from pouring out worries and from receiving sympathetic advice, great benefits ensue.' The cause of the anxiety or mental tension present must be discovered and removed or, if this is not possible, the patient must be persuaded to accept it judiciously.

It is obvious that the principles on which the treatment of hyperchlorhydric dyspepsia are based are identical with those described for the treatment of peptic ulcer (see pp. 269 to 272). Since the symptoms are in general less severe in hyperchlorhydric dyspepsia, and serious complications such as hæmorrhage and perforation do not occur, it is seldom necessary to confine the patient to bed except in the most severe cases, when a few days rest may be of real value. In such cases the purée diet (see Diet No. 10, p. 463) recommended for the first stage of treatment of peptic ulcer should be used, but in the majority of cases treatment can start with the intermediate stage diet (see p. 272).

2. Achlorhydric and Hypochlorhydric Dyspepsia

Achlorhydria may be defined as a condition in which free hydrochloric acid is not found in any fraction of a test meal. The failure of gastric secretion may be permanent and complete, or it may be a temporary state from which entire recovery may result. Pepsin and the hæmopoietic factor may also be absent, the condition then being called achylia gastrica.

Achlorhydria and hypochlorhydria may be constitutional in origin or acquired. Symptoms may be present or absent. Only in the former case is the term achlorhydric or hypochlorhydric dyspepsia justified. The recognition and treatment of the achlorhydric state is of great importance because there is ample evidence to show that it may precede, accompany or result from serious organic disease of the stomach, *e.g.* carcinoma or gastritis. Before the diagnosis of functional achlorhydric dyspepsia is accepted it is necessary to exclude the presence of cancer of the stomach and chronic gastritis. It should also be remembered that achlorhydria is invariably present in Addisonian pernicious anæmia and no infrequently is found in cases of rheumatoid arthritis, toxic goitre and asthma, and in debilitated patients during and after toxic and infective states. If, after a full and careful investigation, no adequate explanation of the achlorhydria or hypochlorhydria i forthcoming, it may be assumed that the condition is constitutiona in origin. It should be remembered that the incidence of achlorhydria in healthy young adults is approximately 4 per cent; by th age of 40 the figure reaches 10 per cent, and by the age of 60 30 per cent.

Since the absence or reduction of free hydrochloric acid in th stomach produces by itself no symptoms, any which may be present, in the absence of organic disease accompanying the achlo hydria, must be assumed to be due to a hypotonic condition of th gastric musculature. This gives rise to feelings of heavines discomfort and weight in the abdomen after meals, or to rapi emptying of the stomach through a patulous pylorus, which lead to symptoms of wind and discomfort, and occasionally to diarrhœ as the result of intestinal hurry.

Experience has shown that the symptoms of functional achlo hydric and hypochlorhydric dyspepsia can often be correlated wit emotional and anxiety states which, when they occur in patien with the hyposthenic diathesis, lead to a reduction in motor ar secretory power.

Treatment. In functional achlorhydric or hypochlorhydr dyspepsia the principles of treatment may be summarized follows: (i) removal or correction of causal factors; (ii) correction faulty habits of eating and drinking (see pp. 261 to 265); (i correction of faulty posture and bad body mechanics, and improv ment of muscular development by postural and breathing exercis and by regular exercise in the fresh air; (iv) the administration of drugs, *e.g.* hydrochloric acid, pepsin, strychnine, preparations of vitamin B_1, and sedative drugs when emotional and anxiety states are present; (v) psychological treatment for the correction of faulty methods of living at home and at work, and removal of causes of anxiety and tension; and (vi) the provision of a suitable diet, which should be bland in character, adequate in Calories, minerals and vitamins, mechanically non-irritating, but chemically stimulating, and given in moderate amounts in a relatively dry state at three or four meals. For the treatment of achlorhydric dyspepsia see Diet No. 11, p. 464.

We would again emphasize that in peptic ulcer, chronic gastritis, and hyperchlorhydric and achlorhydric dyspepsia the most important part of treatment for the prevention of recurrence is the instruction of the patient in correct methods of chewing his food, the avoidance of indigestible articles of food, eating slowly, resting before and after meals, and the regulation of his method of living in such a way that emotional and anxiety states will interfere with the physiology of the stomach to the minimum extent.

DIARRHŒA

Introduction

Diarrhœa may be defined as an abnormally rapid passage of intestinal contents through the alimentary tract resulting in loose or watery unformed stools. It may be acute or chronic. It is necessary to point out that diarrhœa is a symptom, not a disease. Hence treatment must be preceded by a careful investigation of the cause.

The causes may be functional or organic. If functional, the diarrhœa results from overactivity of the neuromuscular mechanism consequent on emotional states or is reflexly caused by disease of adjacent organs. If organic, diarrhœa follows irritation or inflammation of the mucous membrane of the bowel by physical, chemical or bacterial agents. The most important cause of inflammation of the mucous membrane is bacterial infection, as occurs in typhoid fever, dysentery and food-poisoning, while the commonest cause of mechanical irritation of the bowels is the ingestion of excessive amounts of coarse indigestible articles of food, or the passage into the colon of imperfectly digested food constituents, as occurs in intestinal carbohydrate dyspepsia, putrefactive diarrhœa and steatorrhœa. Diarrhœa may co-exist with constipation, and result from the irritation of the mucous membrane by impacted scybalous masses, the fæcal delay being due to hypertonic or atonic states, or organic disease of the lower intestinal tract.

For the recognition of the different types of diarrhœa a carefu history must be taken, and naked-eye, chemical and microscopic examination of the stools made. Where bacterial infection is believed to be the cause, the fæces should be sent to the laboratory for investigation. In other cases, both for purposes of diagnosis and for assessing progress, radiological and sigmoidoscopic examinations may be required. By these means it is possible to differentiate diarrhœa due to functional causes (in which, apar from the looseness of the motions, nothing abnormal will be found, from diarrhœa due to infection or inflammation, in which mucus blood and pus are present. The naked-eye appearance of the stoo may confirm not only these signs of inflammation but may indicate from the consistence of the motions, the probable site of the lesion If the stool is extremely watery, small-intestinal hurry is probabl present. If the lesion is in the colon the stool is usually thicker with a consistency resembling gruel.

A frothy acid stool suggests a failure of digestion of carbohydrate, and a foul alkaline stool indicates a deficient digestio of protein, while a clay-coloured or pale stool favours a diagnosi of steatorrhœa. Microscopic examination should always be undertaken when the above conditions are suspected, to demonstrat whether an excess of carbohydrate (starch granules), protein (undigested muscle fibres), or fat (fat globules and fatty acid crystals is actually present in the fæces.

General Treatment of Diarrhœa

Many patients consult their doctor for diarrhœa, when in realit they are suffering from constipation which they are treating wit purgatives. Accordingly inquiry should be made into this possibility, and if cathartic drugs are being taken they must be stoppe before investigations into the causation and treatment of th diarrhœa is begun.

Where diarrhœa is believed to be due to the ingestion of irritati or toxic material, treatment should be started by giving ½ to 1 c of castor oil. If the diarrhœa is functional in origin, correction the psychogenic factors is of particular importance, and sedati drugs may be required. If the cause is reflex, *e.g.* appendicitis or cholecystitis, operation must be considered.

If the cause is bacterial, and serum or a suitable sulphonamide compound is available, as in dysentery and cholera, these should be given. If infection with *Entamœba histolytica* is suspected, *i.e.* amœbic dysentery, emetine is a specific remedy. If the fault lies in a failure of secretion of hydrochloric acid by the stomach (gastrogenous or achlorhydric diarrhœa), dilute hydrochloric acid, 1 to 2 drachms well diluted with water and flavoured with orange juice, should be taken with meals. If a failure of digestion in the small intestine is the causal factor, the appropriate measures for the treatment of intestinal carbohydrate dyspepsia, putrefactive diarrhœa or steatorrhœa, as detailed below, must be advised.

Treatment of the Acute Stage of Diarrhœa, from any Cause

The principles of treatment are as follows:

1. The prevention and correction of dehydration and hypochloræmia. In very severe diarrhœal diseases such as cholera, it may be necessary to augment the oral administration of fluids by the parenteral injection of normal saline.
2. The elimination of mechanical irritation of the bowel by prescribing a diet which leaves the minimal food residue.
3. The prescription of drugs for allaying inflammation, relieving colic, adsorbing toxins and flatus, and reducing the excitability of the neuromuscular mechanism. For these purposes calcium carbonate, bismuth carbonate, atropine, kaolin, charcoal and opium or its alkaloids may be mentioned.
4. *Diet.* In very severe cases it may be advisable, for a period not exceeding 48 hours, to give the patient nothing by mouth except water, or better still, ½-normal saline (½ teaspoonful sodium chloride to 1 pint of water), flavoured with fruit juice and glucose, augmented if necessary by the parenteral administration of glucose saline. The patient should be allowed to sip small quantities of the fluid as frequently as desired during the waking hours.

In the majority of cases, treatment may start with two-hourly feeds of 5 to 8 oz. of one or other of the following – barley water, albumen water, whey, arrowroot made with water, or glucose lemonade (5 per cent). With the improvement in the patient's condition, which usually occurs within 24 to 48 hours, milk diluted with equal parts of water, junket and custard, fruit and milk jellies, and chicken tea may be added. With further improvement, Benger's or other proprietary invalid foods, soft-boiled or scrambled eggs, refined cereals and milk puddings can be take and within a week the patient will probably have reached the lo residue bland diet recommended for chronic dyspepsia (see pp. 2 and 266).

Treatment of Subacute and Chronic Diarrhœa, such as occurs Inflammatory or Irritative States of the Intestinal Tract, e. Dysentery and Ulcerative Colitis

The general principles of treatment of any chronic diarrhœa are follows:

1. Rest, both mental and physical, must be ensured, and t patient must not be allowed to become chilled.
2. Drugs may be necessary (see Acute Diarrhœa above).
3. Local treatment of the colon by saline and medicated enema is of value at appropriate stages of the disease.
4. A diet must be prescribed which (i) must be adequate Calories, food constituents, minerals and vitamins, particularly the diarrhœa is long continued; (ii) should be given in sm quantities at relatively short intervals; (iii) should be mechanica non-irritating and chemically non-stimulating. For this purp coarse and indigestible articles of food containing cellulose must avoided and condiments and spices prohibited (see p. 265).

The objects to be attained are the prevention or correction toxæmia, exhaustion, dehydration, anæmia, and loss of weig Protein of high biological value (see p. 27), and minerals a vitamins must be supplied in optimal quantities, while foods r in fat and starches should be moderately restricted.

For the majority of cases of chronic ulceration of the colon, well-balanced, nutritious, bland diet low in roughage, as descril on pp. 265 and 266 will be found satisfactory. The addition vitamin concentrates (A, B, C and D) is recommended during active stage of the disease when toxæmia and malnutrition marked. Should the diarrhœa fail to be controlled by the ab measures, it may be advisable to try the effects of the high prot low fat, dextrinized diet as detailed for the treatment of sp (Diet No. 14, p. 466) or the pulped apple diet as recommended Moro (1929) for the treatment of dysentery and colitis. For t purpose 150 to 200 grams of peeled, raw, ripe, grated apple p are given six or seven times daily at intervals of two hours. apple diet should not be continued for more than three or days, as it is deficient in both Calories and essential food fact After this period, a small helping of lean tender steak or white with two rusks or a slice of crisp toast must be given at two of the meals in place of two of the apple feeds, and thereafter the diet must be cautiously built up to the bland nutritious diet described on pp. 265 and 266.

Patients with ulcerative conditions of the colon should remain on a mechanically non-irritating, low residue diet until the diarrhœa has disappeared and microscopical and chemical examination of the fæces for blood and pus is negative. Their return to a normal diet should be carried out carefully and in slow stages, and it is probably wise permanently to exclude foods which are known to be indigestible or which contain excessive amounts of coarse residue (see list on p. 265) from the diets of patients who are known to have had ulcerative colitis or chronic dysentery.

Treatment of Diarrhœa due to a Failure in Digestion

1. *Intestinal Carbohydrate Dyspepsia.* If a patient suffering from diarrhœa, abdominal discomfort and colonic flatulence passes motions which are frothy and strongly acid in reaction, the condition of intestinal carbohydrate dyspepsia should be suspected. The diagnosis will be confirmed by finding in the fæces large numbers of starch granules. Owing to an irritable state of the small intestine, the undigested starch is hurried into the colon, some being fermented with consequent irritation of the colon while some is passed unchanged in the fæces. The treatment consists of the exclusion or reduction of starchy food from the diet. The mono- and disaccharides in the form of glucose, cane-sugar, honey and jam are allowed. Bread, toast, scones, pastry, potatoes, green peas, lentils and root vegetables are either excluded from the diet or reduced in quantity, depending upon the severity of the condition. In the majority of cases a slice of crisp toast or one or two rusks can be taken with safety at breakfast and tea. As the condition improves, starchy foods should be slowly increased. The use of Taka-diastase (Parke, Davis & Co.), a starch splitting ferment, may be of value in enabling additional amounts of starchy food to be included in the diet. In severe cases codeine is of service retarding the small intestinal hurry.

2. *Putrefactive Diarrhœa.* A failure in the digestion of protein the small intestine is indicated by the presence of dark, foul-smelling alkaline stools in which muscle fibres in large numbers, with nuclei and striation intact, may be recognized under the microscope. Under these conditions beef, mutton, chicken, liver and kidney should be prohibited from the diet, the protein content

of which should be obtained from the dairy products milk, cheese and eggs. Cereals and vegetables need not be restricted, as carbohydrate digestion is unimpaired. The Lacto-Vegetarian Diet No. 12 (p. 464) will meet the above requirements. It is in putrefactive diarrhœa that *Lactobacillus acidophilus* milk is of particular value.

3. *Steatorrhœa.* Excess of fat in the stools may be due to an absence of bile from the intestine, due to obstruction of the biliary passages, a failure in secretion of lipase by the pancreas, or defective absorption of fat from the bowel as occurs in tropical and non-tropical sprue (cœliac disease, idiopathic steatorrhœa).

In all forms of steatorrhœa the essential method of controlling the diarrhœa is by reduction of the fat intake. It is best to begin with a diet which is as nearly fat-free as possible, and to add small quantities of fat every few days until the patient's limit of tolerance is reached. The following articles of food are particularly rich in fat and should be rigorously excluded at first.

All fried foods.
All foods made with much butter, lard or margarine, *e.g.* pastries, pies, sauces, suet puddings and cakes.
All visible fats of meat, whether beef or mutton.
Fat meats such as pork, duck, goose, ham, bacon, sausages.
Fat fish such as salmon (tinned or fresh), mackerel, herrings, sardines.
Olive oil, mayonnaise, cod-liver oil.
Nuts and all sweets and cakes containing nuts.
Chocolate and cocoa.
Toffee, caramels and marzipan.
Cream.
Of all fat, milk fat is best tolerated.

The first fatty food to be added to the diet is milk.

For further details of the treatment of steatorrhœa due to *disease of the liver* with obstruction of the biliary tract see pp. 300 to 308.

When the steatorrhœa is due to *disease of the pancreas* (chronic pancreatitis, carcinoma) the dietetic treatment may be extremely difficult, as defects in the digestion of carbohydrate and protein may also be present. Nevertheless it is from these two food constituents that the diet has mainly to be constructed, because the principal cause of the diarrhœa is the steatorrhœa. Hence all food rich in fats must be prohibited, *i.e.* butter, cream, bacon, suet and fatty meat and fishes. Milk should be given skimmed, although a small quantity of fresh milk may be allowed if previously peptonized. Small amounts of crisp toast, rusks and cereals are permitted. A small helping of chicken, white fish or very lean meat is allowed at lunch and dinner, with any vegetable (except potatoes or pulses). Fruits (fresh or stewed), jams, jelly and sugar should be taken plentifully. Diet No. 13 (p. 465) will be found suitable for the initial treatment of chronic pancreatic deficiency. The deficient secretion of the pancreas may be helped by substitution therapy. Proprietary preparations containing the pancreatic enzymes are available in tablets or capsules covered with a protective coating resistant to the hydrochloric acid secreted by the stomach – *e.g.* Festan (Bayer), Panacoids (Reed and Carnrick). Two tablets or capsules should be taken three times a day before meals.

When the fatty diarrhœa has been brought under control, the caloric content of Diet No. 13 must be increased gradually by allowing larger helpings of fish, chicken and lean meat, and by including, cautiously, additional amounts of fat in the form of fresh milk and small amounts of butter or vitaminized margarine.

In *sprue* and *idiopathic steatorrhœa* the rationale of treatment is simpler, as the main feature of the disease is a failure in absorption and not in digestion.

The objects of treatment are the restoration of function of the alimentary tract, and the correction of demonstrable deficiencies. The principles of treatment may be summarized as follows: (i) adequate rest in bed, and warmth; (ii) the administration of calcium, iron, and liver or liver extract for the correction of tetany, hypocalcæmia and anæmia; (iii) the administration of preparations of vitamins A, B and D; if a hæmorrhagic state is present, vitamin K therapy should be given, as recent research suggests that in these cases its cause lies in a deficiency of blood prothrombin; and (iv) the prescription of a diet which is mechanically non-irritating, high in protein, and low in fat and carbohydrate, as recommended by Hamilton Fairley. The ratio of protein : fat : carbohydrate = 1·0 : 0·3 : 1·3 instead of the normal ratio of 1 : 1 : 5.

The degree of carbohydrate restriction will vary from case to case, and can be assessed clinically by the symptoms of abdominal discomfort and distension and by the amount of frothy fermentation and the acidity of the stools. This dietetic regime has largely superseded the milk diets, raw meat diets and fruit diets which have been recommended in the past. The protein can be given in the form of lean red meat, lightly cooked, or as milk protein in the form of a dried, defatted milk powder, as prepared by Cow and Gate and marketed under the name 'Sprulac'. In 'Sprulac' the ratio of protein : fat : carbohydrate = 1·0 : 0·3 : 1·0. This prepar tion is particularly valuable for the treatment of steatorrhœa in th tropics, where tender red meat or a supply of good milk is n always available.

The instructions about the restriction of foods rich in fat, described for the treatment of diseases of the pancreas, apply wit equal force to the treatment of sprue and idiopathic steatorrhœ

Articles of food which are mechanically irritating or chemical stimulating, as outlined on p. 265, should be avoided; vegetabl must be passed through a fine sieve and made into purées, a starchy foods such as potatoes, cereals and bread must be tak in very small quantities at first but can steadily be increased amount with improvement in the patient's clinical condition. F the preparation of the meat, the best quality of lean rump ste should have all the gristle and fat removed and, after fine mincir should be lightly cooked in a dry pan for two or three minut without the addition of any grease or water.

The graduated high-protein meat and high-protein milk di shown in Diets Nos. 14, 15, and 16 (pp. 466 to 468) those constructed and recommended by Hamilton Fairley. T rapidity with which the diets are changed is determined by t progress of the patient's illness, indicated by the disappearance intestinal flatulence and abdominal distension, and by the ch acter of the stools.

In our experience a high protein, low fat diet is best attain and the diet is much more palatable to the patient, if 3 to 4 oun of lightly cooked, finely minced steak are given at breakfast, lu and dinner with three intermediate feeds of 2 ounces of Spru It is desirable on one day to substitute lightly cooked, ten liver for one of the meat meals, and on the next to give, in addit to one of the meat meals, a soup made from half a pound of liv At present it is not known whether the protein of beef-steak superior to the protein of fish in the treatment of sprue idiopathic steatorrhœa, but in the writers' experience the repl ment on occasion of the breakfast or dinner meat ration by equivalent quantity of boiled or steamed white fish is justifia and is much appreciated by the patient.

For a discussion of the treatment of anæmia in sprue idiopathic steatorrhœa see p. 383.

CONSTIPATION

For comfort and health, most people require a daily bowel movement. However, since good health may be maintained by persons who defæcate only once every two or three days, it is unwise to insist that a daily bowel motion is essential in every case. The ill effects of constipation have probably been exaggerated; nevertheless evacuation which is insufficient and infrequent leads to definite symptoms such as malaise, headache, coated tongue, foul breath, and mental sluggishness.

The harmful effects of constipation are often ascribed to the absorption of toxins from the fæcal material in the colon. Evacuation of the bowels gives such prompt relief from symptoms that such a cause is unlikely. A more probable explanation is that the pressure of the fæcal mass on the nerve-endings in the colon and rectum initiates afferent impulses which lead to the symptoms characteristic of the overloaded intestine. The treatment of constipation by purgatives, leading to inflammation of the intestinal mucosa and the too rapid entry into the colon of fluid small intestinal contents, is much more likely to cause the so-called auto-intoxication than constipation itself.

Constipation may be manifested in three ways: 1. defæcation may be infrequent; 2. the stools may be inadequate in amount; 3. the stools may be abnormally hard and dry.

Delay in the passage of the fæces may occur:

1. *In their General Movement along the Large Intestine.* – *Colonic Stasis.* The general movement of the fæces along the intestinal canal is purely involuntary, and is dependent on an adequate muscular power of the intestinal wall, properly stimulated to peristaltic activity by suitable food residue. Colonic stasis may be due to (i) *weakness of the musculature of the intestinal wall*, leading to ineffective propulsion of the fæcal contents along the colon into the rectum. The weakness may be constitutional in origin or more frequently it develops in later life as a result of old age, debilitating disease, pregnancy, or the long-continued ingestion of a faulty diet. This type of constipation is often called *atonic* constipation. (ii) *Spasm of the colon*, leading to a retardation of the forward movement of the intestinal contents – *spastic* constipation. (iii) *Abnormal consistency of the fæces.* To permit of easy expulsion, he colonic contents must be semi-solid, and to produce fæces of this character the food must contain a sufficiency of cellulose. Concentrated foods which are almost completely digested and

absorbed give a fæcal mass which is small, hard, and difficult of propulsion. The taking of insufficient amounts of fluid daily is another important factor leading to the formation of scybalous fæces.

2. *In their Evacuation from the Pelvic Colon and Rectum—Dyschezia.* The desire to defæcate is caused by the entry into the rectum of the fæces which have accumulated in the pelvic colon during the preceding twenty-four hours. The passage of the fæces from the pelvic colon to the rectum is brought about by a mass peristalsis of the colon, this being stimulated by the entry of food into the stomach (gastro-colic reflex) or by the morning activities in getting up.

Dyschezia may be due to:

(i) Faulty habits with regard to time or regularity of going t stool.

(ii) Depressed reflex sensitivity. If the subjective desire fo defæcation is habitually neglected there is a lessening in the inten sity of the sensory impulses set up when fæces enter the rectum This is also found in diseases involving nervous depression, *e.g* neurasthenia.

(iii) Weakness of the voluntary muscles of defæcation (th diaphragm, the abdominal muscles, and the muscles of the pelvi floor).

Treatment of Constipation

Although it is advisable to consider the treatment of aton and spastic constipation separately, there are certain comm principles which are applicable to the prevention and treatme of fæcal stasis in general.

1. *The Prevention and Correction of Faulty Habits.* The basis constipation is frequently laid in infancy and childhood by lack insistence on the part of the mother on a regular time-table f going to stool. The same lack of training may arise in scho children or in adults from a failure to answer nature's call wheth due to laziness, hurry or lack of sufficient lavatory accommodati Once the reflex resulting from the entrance of fæces into t rectum is lost, it can only be regained with difficulty by persist and unhurried attempts to move the bowels at the same ti every day whether the desire to defæcate is present or not. T patient must also establish a correct attitude of mind towards t problem. The influence of worry, anxiety, alteration of occupat and fatigue are factors which may affect the normal rhythm

evacuation. This must be pointed out to the patient, but it is equally important to reassure him that the evil effects of constipation have been exaggerated, and when present are due more to the taking of purgatives than to the constipation itself.

2. *The Taking of adequate Physical Exercise.* This is an important factor in treatment, and applies equally to the obese, flabby, atonic person and the thin, visceroptotic individual with faulty body mechanics, an anxiety neurosis and a spastic colon. Regular exercise (walking, cycling, riding, rowing, golfing, etc.) in the open air, supplemented if necessary by 'Swedish exercises' for the development of the abdominal muscles, should be recommended and graded according to the age and physical state of each individual.

3. *Diet.* The foods which are of particular value in the treatment of constipation are:

(i) Those which contain adequate amounts of cellulose. Since this substance is not fully digested by enzymatic or bacterial activity, it enters the colon in a semi-solid form and initiates peristaltic activity by its bulk and by its mechanically irritating properties. The refining of cereals by milling, with the resultant loss of the valuable husks, has been a major disaster for the poorer sections of the community owing to the loss of vitamins (especially B), minerals and roughage. Therefore in prescribing a diet for constipation, wholemeal cereals should replace the more finely milled cereal products. The leafy vegetables (cabbage, spinach, lettuce, etc.) and the roots (carrots, turnips, onions, etc.) are also valuable sources of roughage and vitamins. It is unwise to prescribe initially an excessive amount of vegetables to a constipated patient who has been accustomed for years to a low residue diet, as the colon cannot be expected to adjust itself suddenly to such a rapid change. At first, vegetables should be given in moderate quantities at two meals and should be sieved and served as purées. In the spastic form of constipation it may be necessary to continue with the sieving of vegetables for months or even permanently, but in atonic constipation the quantities given should be progressively increased, and a coarser sieve used, until the vegetables can be taken raw or cooked in the ordinary way without sieving.

(ii) Fruit, fresh and cooked, is of great value, containing as it does roughage in the form of cellulose and also organic acids which possess laxative properties. The quantity eaten daily should be increased gradually until a good serving is taken at two meals a day at least. In the initial stage of treatment of atonic constipation and throughout the treatment of the spastic variety, it is ofte desirable to exclude raw fruit such as apples and pears and to rel on cooked fruit which has been sieved, fruit juices and fruit jellies As with vegetables, an attempt should be made to increase steadil the quantity of raw fruit eaten and to desist from the use o sieving subsequent to cooking.

(iii) Fats are useful in the treatment of constipation. They pr duce their laxative effect by increasing the output of bile and b a stimulating effect of the fatty acids on the intestinal mucos When constipation is associated with obesity, however, fat in th diet must be restricted.

(iv) Animal experiments suggest that the tone of the intestin muscles is closely associated with the intake of aneurin (vitam B_1). The ingestion of adequate amounts of green vegetables a wholemeal cereals will ensure a good supply of this vitami Nevertheless, we believe that an extra supplement of vitamin in the form of Bemax, Marmite or yeast tablets is of real value the treatment of constipation.

(v) Reference has already been made to the importance of taki plenty of fluid to help to keep the fæces soft; 6 to 8 glasses of flu should be taken in the 24 hours. The great value of the glass of h water before breakfast has long been recognized. When t stomach and small intestines are empty, the water passes rapi down to the colon and excites the peristaltic movements.

Diets for the treatment of atonic constipation incorporating t above principles will be found in Diets No. 17 and 18 (pp. 469 a 472).

4. *Drugs*. For the treatment of chronic constipation relia should be placed essentially on the correction of faulty habits a the prescription of a proper diet as described above; drugs sho play only a minor role. Cathartics such as castor oil and calo are contra-indicated, but it may be necessary to use mild vegeta laxatives (senna, cascara sagrada, etc.) for a period in patients whom constipation has been present for years. Inert substan which lubricate the bowel content (liquid paraffin) or add n irritant bulk to the fæces (agar-agar, psyllium) are often of gr value.

Spastic Constipation and Muco-membranous Colic

In certain individuals suffering from spastic constipation, bout diarrhœa alternate with constipation, and in some cases shred mucus or mucous casts of the bowel are passed. Such patients often said to be suffering from mucous colitis. However, as no evidence of inflammation can be found by sigmoidoscopic examination or microscopic investigation of the fæces, the term 'mucous colitis' is not justified. Sufferers from muco-membranous colic are almost invariably unstable and emotional middle-aged women who gradually develop a 'constipation complex', and are inveterate takers of cathartics. The colon in such patients is in an irritable state; it can be palpated in a state of spasm through the abdominal wall, and the spastic condition can be confirmed by radiological examination.

The essential principles of treatment of spastic constipation are:

1. Psychotherapy.

2. The use of sedative and antispasmodic drugs (phenobarbitone and belladonna).

3. The cessation of purgatives and colon lavage.

4. The use of a bland, highly nutritious diet, low in roughage and rich in vitamins. It is essential to reduce the roughage, initially at least, as its presence would only irritate the colon and increase spasm. The fæcal bulk necessary to promote peristalsis should be obtained from agar-agar, a product derived from seaweed (two heaped teaspoonfuls of powdered agar thrice daily after meals) or from the vegetable mucilages (*e.g.* psyllium), while liquid paraffin is of great value as a lubricant. An attempt should be made at suitable intervals gradually to increase the intake of fruit and vegetables. The above principles are incorporated in Diet No. 19 (p. 472).

CHAPTER XIII

DISEASES OF THE LIVER AND BILIARY TREE

HEPATITIS AND CIRRHOSIS OF THE LIVER

THE term hepatitis should strictly be limited to a diffuse inflan
mation of the tissues of the liver, but it is generally extended
cover, in addition, the resulting reactions, degenerative or repar
tive, which follow the injury. It is thus possible to include und
the term hepatitis: (1) acute and subacute yellow atrophy of t
liver (severe acute and subacute hepatitis or hepatic necros
which may occur during the course of the toxæmias of pregnan
and certain infectious diseases such as pneumonia, amœb
dysentery or spirochætal jaundice, etc., or from toxic drugs a
chemical poisons (such as arsphenamine, chloroform, phosphor
cinchophen and trinitrotoluene); (2) infective hepatitis, former
called simple catarrhal jaundice (mild acute hepatitis); and
cirrhosis of the liver (chronic interstitial hepatitis).

When acute hepatitis is so severe as to be termed 'acute yell
atrophy of the liver', recovery seldom occurs, death resulting fr
hepatic failure, erroneously called 'cholæmia'. When hepatitis
moderate or mild, recovery may be complete or partial. In
latter case the disease passes into the state of chronic hepat
(cirrhosis) which may, however, arise insidiously from oft-repea
slight damage to the liver from toxic substances, bacterial
chemical, or from dietary deficiency. In the chemical gr
alcohol is the most important cause.

PRINCIPLES OF TREATMENT

In all types of hepatitis the first step is to search for and, if possi
remove the injurious factor causing the condition. Dietetic tr
ment of hepatitis will depend on: (1) the acuteness and seve
of the condition, and (2) the presence or absence of jaundice.
acute and subacute hepatitis, owing to damage to the pa
chymal cells of the liver and obstruction to the bile canali
jaundice of varying degree is present. In chronic hepatitis
rhosis of the liver) the regeneration of liver cells and small
ducts, except in the terminal stage of the disease, is usuall
sufficient magnitude to prevent the occurrence of jaundice.

It will also be remembered, when considering the principles of treatment, that the liver is intimately concerned in the metabolism of fats, carbohydrates, and proteins, the storage of vitamins, the excretion of bile, and the neutralization of certain toxic substances, either metabolic or introduced from without.

THE PROTEIN INTAKE IN HEPATITIS

It has long been accepted in clinical practice that a diet rich in carbohydrate protects the liver from hepatotoxic agents, while a diet rich in fat increases the liability to injury. These dietetic principles were founded essentially on the experiments of Opie and Alford, carried out in 1914, which showed that an ample supply of carbohydrates protected the livers of dogs from chloroform poisoning. Until recently it has been the clinical practice to restrict the intake of protein in the diet rigorously with the object of 'sparing' the liver, since the metabolism of protein derivatives requires hepatic activity. There are physiological, experimental, and clinical grounds for believing that restriction of protein is contra-indicated.

Physiological

(1) It was formerly believed that deamination of amino-acids occurred only in the liver. Recent physiological research suggests that the liver is neither the sole nor possibly even the most important organ to undertake this work. The formation of urea from ammonia undoubtedly constitutes an important function of the liver, but both experimental and clinical evidence indicate that this is probably the last function of the liver to be seriously affected by disease or injury.

(2) Since cellular repair depends essentially on the protein components available, the belief that a defective intake of protein in the diet can be compensated for by a high intake of carbohydrate violates fundamental physiological principles. If the intake of protein is insufficient, the requirements will be met by a break-down of endogenous protein with resulting wasting of the patient.

(3) The liver plays a leading part in the manufacture of the plasma proteins and is an important storehouse of protein readily available for distribution. It has long been known that hypoproteinæmia, with its resultant œdema, commonly occurs in hepatic disease, especially in the more chronic variety called cirrhosis of the liver. To reduce the intake of protein in the diet

in these circumstances is obviously undesirable. A similar physiological outlook long permeated views on the dietetic tre ment of chronic and subacute renal disease, in which albuminu was accepted as an indication for reducing the intake of protei

(4) While choline and methionine are of particular value for t removal of fatty acids from the liver, thus preparing the way cellular repair, proteins rich in nucleo-protein are of especial i portance for regenerative effects. For this purpose liver as article of diet has been strongly recommended.

Experimental

(1) There is ample evidence that fatty degeneration of the li may precede cellular necrosis. Experimental cirrhosis of the li has been produced in animals both by a high fat diet or a protein diet. It is interesting to note that an excessively h carbohydrate intake, if given for long periods with inadequ protein, may cause fatty changes to develop, resulting from conversion in the liver of carbohydrates to fat.

(2) A high protein diet will protect animals against hepatot agents such as arsenical compounds, chloroform, carbon te chloride, etc., while a low protein diet facilitates hepatic inj Whipple showed that hypoproteinæmia in dogs, produced venesection, made their livers more susceptible to toxic age including chloroform.

(3) Glynn and Himsworth (1944) have shown that if rats kept on a diet low in protein, pathological changes in the result which are similar to those of acute and subacute he necrosis in man. These effects can be prevented by the add of casein or methionine to the diet but not by choline.

McCallum and Miles (1945) injected material from human of infective hepatitis into rats which were receiving a diet defi in protein. The dietary deficiency, however, was not by sufficiently severe to produce hepatic necrosis. After se passages a fatal hepatic necrosis was produced in the anima the protein-deficient diet, although no pathological effects be produced in controls which had previously been receiving adequate in protein.

It should be noted that the value of high protein die supplements of methionine in the animal experiments q above, are essentially prophylactic in character. That is t there is ample proof of their value in the prevention of h disorders in animals which have been depleted of protein.

factory evidence is lacking, however, that a similar beneficial effect can be secured in animals whose protein intake was adequate before the production of the hepatic injury. In fact, the administration of excessive quantities of protein during the initial stage of severe acute hepatitis may actually be harmful: thus, Bollman and Mann (1930) showed that dogs whose livers had been damaged by ligating the common bile duct or by poisoning with carbon tetrachloride developed ascites and liver failure more readily on a high than on a moderate protein intake.

Clinical

(1) Patek and Post (1942) treated 54 patients with chronic hepatic diseases with a highly nutritious diet containing a generous allowance of protein (149 grams daily) with supplements of brewer's yeast (50 grams daily), and liver extract twice weekly. After two years of treatment 45 per cent of the treated group were alive as compared with 21 per cent of a control series not receiving the high protein, high vitamin diet. In addition some cases showed a complete disappearance of œdema and jaundice, while the plasma proteins and liver function tests approached or reached normal values. Treatment had to be continued for many months before such beneficial effects were produced.

(2) Butt and Snell (1942) also treated 50 cases with a similar diet and record encouraging results.

(3) Fagin and Zinn (1942) treated 5 carefully controlled cases of cirrhosis of the liver with a solution of amino-acids given intravenously, and noted a marked diminution in size of the liver and spleen, a reduction of œdema, and an improvement of findings in tests for hepatic insufficiency.

(4) Ravdin (1943) made biopsies of livers from patients with histologically normal organs and from patients with severe hepatic disease, some of whom had received a high protein diet and others low protein diet before operation. Chemical assay clearly indicated that the fatty-acid content of the livers of patients on a low protein diet was exceedingly high but was normal in those receiving a high protein diet prior to operation.

(5) Beattie and Marshall (1944), who have treated large numbers of cases of hepatitis, particularly of the type occurring some ninety days after treatment with arsphenamine, are convinced of the value of a high protein diet.

(6) Hardwicke (1945) carried out an investigation on two groups of young airmen suffering from infective hepatitis who were

given high and low protein diets respectively. One group of 35 airmen received 180 grams of protein daily, while the other group of approximately the same number received 50 grams daily. It must be emphasized here that the experiment was not designed to ascertain quantitatively the optimal intake of protein in hepatitis, but rather to test the validity of the hypothesis that recovery from liver damage of toxic or infective origin is hastened by increasing the patient's dietary protein intake. For this reason, two extremes of protein intake were chosen. In both groups the carbohydrate intake was high and the fat intake low. No difference between the two groups was found in regard to the course of the illness as judged by clinical and chemical standards. This finding is perhaps not surprising for two reasons:

(1) The patients were healthy young adults who for long periods before their illness had been taking a well-balanced and ample diet containing more than 100 grams of protei daily. Accordingly it can be safely assumed that they had ample reserve stores of protein available.

(2) Infective hepatitis has a very low mortality rate in health young adults (approximately 1 in 1,000), and probably a least 95 per cent of cases proceed spontaneously to complete resolution.

With such small numbers as were concerned in Hardwicke' experiment it would be unreasonable to expect statistical proo of the value of any form of treatment on the course of the disease. Of much greater importance than the negative finding reported is the positive evidence that throughout the course infective hepatitis, and during the acute stage with fever an jaundice, the patients were able to eat and metabolize 180 gran of protein daily without any deleterious effects. We know of n benefits which would result from such a high intake of protein infective hepatitis, whereas the danger undoubtedly exists that the damage to the liver were sufficiently severe, harmful resul might occur. Similarly, we would not advocate such a low inta of protein as 50 grams daily except for the initial stage of trea ment for very ill patients. This figure only exceeds slightly t minimum protein requirements for a normal person, while it universally recognized that the needs of the body for protein a increased whenever the basal metabolism rises. This is particula the case where fever and hepatic dysfunction occur togeth Accordingly we feel that the protein intake for infective hepati recommended below (80 to 120 grams daily), which was selected us before the publication of the investigation reported above, is based on sound physiological and clinical considerations.

From what has been said in the preceding sections, it will be apparent that an adequate supply of protein is required for the prevention and treatment of hepatitis. Further, there is much evidence to suggest that the substance in protein on which the integrity of hepatic function and structure depends is methionine. It is proposed therefore to deal with this problem in some detail.

THE IMPORTANCE OF CHOLINE AND METHIONINE IN HEPATIC FUNCTION

Choline

Best and his collaborators proved conclusively that choline (trimethyl-hydroxyethyl-ammonium hydroxide), one of the constituent parts of the lecithins, prevents the accumulation of fat in the liver which occurs when certain diets, deficient in choline, are given to experimental animals. Such deficient diets produce fatty degeneration of the liver followed by hæmorrhagic necrosis, and these lesions do not appear when pure choline is added to the diets. This is the so-called lipotropic action of choline. It was soon afterwards found by Best and by Channon that a sufficient ingestion of casein would also prevent deposition of fat in the liver although the diet contained insufficient choline; and then Channon and his co-workers demonstrated that the amino-acid methionine, present in casein in high concentration, is a lipotropic agent.

Methionine

Methionine (γ-methylthio-α-amino butyric acid), is one of the essential amino-acids, *i.e.* one of those whose presence in the diet is indispensable (see p. 25). Its metabolic importance appears to lie in the availability of its methyl group for the synthesis of methylated substances (*e.g.* choline and others concerned in detoxication processes carried out in the liver), and in the availability of its sulphur atom for the synthesis of the important amino-acid cysteine.

It is clear that, although choline is probably the direct lipotropic agent, preventing or curing fatty infiltration of the liver, the substance of fundamental importance in the prevention and treatment of hepatitis is methionine. For, whereas choline can be synthesized in the body, given an adequate supply of methionine, choline cannot produce new methionine (since homocysteine does not seem to occur in food protein) but can only 'spare' methionine. The importance of this lies in the fact that the methyl group of methionine is required for other methylations, certainly including the synthesis of creatine and probably including various detoxicating processes, in which choline cannot directly participate. Moreover, methionine can act as a precursor of cysteine, which itself has fundamentally important functions, while choline has no such power.

The Clinical Use of Methionine

With such strong evidence available on the importance of methionine in the prevention of experimental hepatitis, it was natural that trials should be made of its therapeutic effect on the clinical condition in human beings. Beattie (personal communication) believes that the administration of 5 to 10 grams of methionine daily by mouth, or by intravenous infusion if vomiting is present, may be a life-saving procedure in patients with acute and subacute hepatic necrosis.

Peters and his co-workers (1945) also claim moderate benefit in post-arsphenamine hepatitis from the administration of both cysteine and methionine. Their claim is supported by independent statistical analysis of their figures. In contrast to these views mention must be made of the report of Turner and his co-worker (1944) who, in America, have treated large numbers of cases o homologous-serum hepatitis with methionine and choline with n obvious beneficial effect. Likewise, the British workers Wilson Pollock and Harris (1945) and Higgins, O'Brien *et alia* (1945 treated series of cases of infective hepatitis with a diet adequat in protein and low in fat to which was added 5 grams of methionin daily. Patients on a similar satisfactory diet but without th addition of methionine were used as a control series. Neither tea of observers was able to find any significant benefit from th supplement of methionine as judged by clinical or chemical criteri

In view of these contradictory reports it is obvious that, at th time of writing, it would be unwise to recommend the routi administration of methionine to cases of hepatitis, especially as th pure substance is in short supply and is extremely expensive.

THE VITAMIN B COMPLEX IN HEPATIC DISEASE

The resistance of the liver to hepatotoxic agents appears to related to the intake of certain vitamins. Thus, a diet defici in the vitamin B complex can produce cirrhosis of the liver in animals. But whereas the addition of the complex in the form of yeast can prevent its production, the administration of aneurin, riboflavin, or pyridoxin in the pure state may fail to achieve this effect.

Although we are unaware of any published evidence which proves conclusively that hepatitis in man can be influenced beneficially by the ingestion of any members of the vitamin B complex, nevertheless, on general principles and because of the experimental results described above, we recommend that yeast should be prescribed. Food yeast would appear to be eminently suitable for this purpose for the reasons described on p. 49.

CONCLUSIONS

The new conception that cirrhosis of the liver may be conditioned by a deficiency of certain dietary factors is of great interest (Glynn and Himsworth 1944). Such a conception might explain how the administration of alcohol to animals on a normal diet fails to produce cirrhosis, although the association of alcoholism and cirrhosis in man is universally accepted. In man the associated defective intake of food which so frequently accompanies alcoholism, together with the consequent impairment of digestion and absorption, may prepare the soil by inducing changes in the liver and by reducing its inherent toxiphylactic properties. It throws light on the frequency with which cirrhosis of the liver occurs in natives of Eastern countries who do not take alcohol but whose diet, particularly in respect of first-class protein and vitamins of the B complex, is notoriously defective. While the full bearing of these experimental and clinical findings on the treatment of hepatitis still awaits final evaluation, the conclusion appears to be inescapable that the old teaching, which prescribed a rigid restriction of protein as essential in the dietary treatment of hepatitis, was based on unsound physiological principles.

On the present experimental and clinical evidence the authors believe that for the prevention of hepatitis and for its treatment, except in the very acute stage, patients should be given a diet containing an ample amount of proteins, especially those, such as casein, which are rich in methionine, and that this diet should be supplemented by a generous intake of vitamins, especially of the B complex. For the reasons stated above, the routine administration of methionine cannot be recommended at the time of writing.

TREATMENT OF HEPATITIS

There exists a considerable divergence of opinion as to the place of a high intake of protein in the treatment of hepatitis. This is probably due to a failure to appreciate that treatment must be regulated according to the stage of acuteness of the pathological process. Thus, in the initial stage of treatment of acute severe hepatitis, when the parenchymal cells of the liver are widely damaged, treatment consists primarily in supplying glucose, fluid and only a moderate quantity of protein. Although the patient is in negative nitrogen balance, it is not possible at this stage to restore the balance by giving large quantities of protein or of amino-acids, since the damaged liver is unable to utilize them for repair or protein synthesis, the excess being merely excreted in the urine as nitrogen. The additional strain on the liver may actually lead to an increase of hepatic damage. It is therefore advisable to restrict the intake of protein to approximately 50 grams in the form of milk protein or casein hydrolysate until th liver has recovered sufficiently to deal with the synthesis an deamination of amino-acids. It is during this initial acute stag that the administration of pure methionine would appear to b particularly indicated, otherwise more protein than the badl damaged liver can metabolize would have to be ingested in orde to supply a sufficiency of the amino-acid. For the reasons given o p. 298 we are unable to recommend at the time of writing th routine administration of methionine.

In contradistinction to severe acute hepatitis, cases beginni subacutely or mildly, or cases of chronic hepatitis (cirrhosis of th liver), can be given a high-protein, high-casein diet from the firs since in such cases the liver is able to utilize the amino-acids a can thus correct the negative nitrogen balance and the hyp proteinæmia so frequently present. The advantage of giving considerable part of the protein in the form of casein is that it not merely an excellent source of all the essential amino-acids b contains methionine in a higher concentration than any ot protein.

Bearing these points in mind it is possible to construct di suitable for the prevention of hepatitis and for the treatment the various types. For both purposes it is recommended that diet should be low in fat and high in carbohydrate; and, except the very acute stage, high in first-class proteins which are g sources of methionine and choline. Vitamin supplements in form of yeast should also be given. The degree to which the restriction of fat should be carried will vary according to the severity of the hepatitis and according to the presence or absence of jaundice. Thus, in severe acute hepatitis, or when jaundice is present, the diet should contain 20 grams or less of fat daily. When such low-fat diets are prescribed, supplements of the fat-soluble vitamins should be given. In chronic hepatitis (cirrhosis of the liver), when jaundice is absent, the restriction of fat need only be moderate, say 60 grams of fat daily, provided the lipotropic effect of methionine is secured by an adequate intake of casein in the form of milk, skimmed milk, skimmed milk powder and cheese. In a chronic disease such as cirrhosis of the liver, it is most desirable to prescribe a diet which is more palatable, less monotonous and richer in the fat-soluble vitamins than the low-fat diet recommended for acute hepatitis. When hypoproteinæmia, œdema and ascites are present, transfusion of plasma may be indicated, but reliance should be placed mainly on prolonged feeding with a high protein diet. Since catarrh of the stomach and intestines frequently accompanies hepatitis, meals should be small in bulk and given at short intervals and should be mechanically non-irritating and chemically non-stimulating (see p. 265).

Acute Severe Hepatitis

In acute severe hepatitis (acute or subacute yellow atrophy or hepatic necrosis) the patient is gravely ill. Nausea is present and vomiting may be serious. Dehydration and acidosis develop and the patient may pass into a state of drowsiness or delirium. In such severe cases it will generally be found advisable to prescribe for a few days an entirely fluid diet of at least 5 to 6 pints daily, given in small feeds at hourly intervals. To secure a high intake of carbohydrate, orangeade or lemonade sweetened with glucose or lactose should be drunk freely. If possible 200 to 300 grams of sugar should be taken every 24 hours. The protein intake should be limited at first to about 50 grams daily. This should be given in the form of fresh skimmed milk or skimmed milk reconstituted from dried skimmed milk powder. Reconstituted skimmed milk may be made of double strength if so desired. Its digestion can be facilitated by predigestion with one of the commercial preparations of pepsin or trypsin. Two and a half pints of skimmed milk daily will supply 50 grams of protein, 40 grams of which consist of casein. A pint of skimmed milk contains approximately 16 grams of casein and 1 oz. of dry skimmed milk powder 8 grams. As previously mentioned, casein is the food protein richest in methionine.

When the condition of the patient is so grave that he can neither ingest nor retain sufficient fluid, glucose and skimmed milk, oral treatment must be replaced or temporarily supplemented by feeding through the stomach tube or by parenteral administration. For the latter purpose 10 per cent glucose in saline or water may be given intravenously by the continuous drip method. Skimmed milk in the fresh state (or reconstituted from dried skimmed milk powder) or casein hydrolysates can be given by gastric or duodenal tube. The quantity given during the initial stage of treatment should not exceed 50 grams of protein or casein hydrolysate daily. Within a week the majority of patients who are going to recover will show definite clinical improvement as indicated by reductio in the size of the liver, accompanied by decrease in nausea an vomiting and a rapid improvement in appetite. Treatment shoul now be started on the lines described below for cases of subacut or mild hepatitis.

Subacute and Mild Hepatitis (Infective Hepatitis, Catarrha Jaundice)

In subacute and mild hepatitis, the dietetic principles incorporate in diet sheet No. 20 (p. 474) can be put into operation. Even i such cases the fluid diet described above may be required for tw or three days if nausea and sickness are prominent features. Di 20 contains 400 grams of carbohydrate, 80 grams of protein ar 20 grams of fat. At least half of the protein should be obtain from skimmed milk and skimmed milk powder. As soon as possib the protein should be increased to at least 100 to 120 grams dai by giving additional helpings of lean meat, liver, chicken and fis Additional carbohydrate foods should be given up to the patien tolerance, since clinical experience has shown the importance the caloric value of the diet being as high as possible. The content of the diet should not exceed 50 grams daily. Diet No. (p. 477) will be found suitable. The diet should be supplement with 30 grams of dried yeast daily (see p. 49).

With further improvement in the patient, the fat intake may increased to 60 to 70 grams as indicated in the section below, devo to chronic interstitial hepatitis. As long as there is any clinica chemical evidence of hepatic derangement, the fat intake should be increased above this level. Patients on a fat intake of 40 gr or less daily should receive a supplement of vitamins A and

Chronic Interstitial Hepatitis, without Jaundice (Cirrhosis of the Liver)

The general principles enumerated above are applicable to cirrhosis of the liver. When hypoproteinæmia, œdema and ascites are present, the protein content of the diet should be raised to 140 to 150 grams by increasing the size of the helpings of protein food. The absence of jaundice and the chronic nature of the disease warrant some relaxation of the severe restriction of fat. Accordingly a daily intake of 60 to 70 grams of fat may be safely allowed, so long as the danger of producing fatty infiltration of the liver is prevented by an adequate intake of casein and yeast. Diet No. 21 (p. 477) will meet these demands if 1¾ oz. of grilled bacon or equivalent (see p. 446) are added to the diet.

OBSTRUCTIVE JAUNDICE

It is necessary to distinguish jaundice due to obstruction of the main bile passages by stone, stricture or malignant disease, from the jaundice occurring in acute hepatitis resulting from cellular damage and intrahepatic obstruction. In the former, the liver cells are at least for some time relatively uninjured, and hence if the obstruction is removed in time no permanent cellular damage will occur. Nevertheless, it is advisable for reasons of safety to ensure that the patient takes an ample supply of sugar and of methionine in the form of skimmed milk and skimmed milk powder. This is particularly desirable as a pre-operative measure to prevent the toxic effects of general anæsthesia. Since jaundice is present, a low intake of fat is indicated. Treatment therefore consists in (i) the relief of the obstruction, either by medical treatment (heat to the abdomen, sedative and anti-spasmodic drugs, and ample fluids) or by surgical intervention; and (ii) the prescription of a suitable diet. When the obstruction is suddenly produced – as by impaction of a gallstone – colic, vomiting and fever may be the presenting symptoms, which necessitate a fluid diet on the lines suggested for acute hepatitis (p. 301). When the acute symptoms subside, or if the obstructive jaundice develops slowly as the result of stricture or of pressure on the common bile duct, a diet containing 300 to 400 grams of carbohydrate, 80 grams of protein, and 20 to 30 grams of fat will be found suitable. Diet No. 20 (p. 474) will meet these requirements. With improvement in appetite and reduction in the jaundice, the protein should be raised to at least 100 grams daily. Diet No. 21 (p. 477) will be found suitable.

CHOLECYSTITIS, CHOLANGITIS AND CHOLELITHIASIS

1. Acute Cholecystitis

The treatment of acute cholecystitis is, at the onset, essentially that of any acute febrile illness. The patient must be kept at rest in bed, heat should be applied to the gall-bladder region and large quantities of fluids should be drunk. So long as the gall-bladder is acutely inflamed it is advisable to keep the organ as far as possible at rest To this end fat, which causes contraction of the gall-bladder should be excluded from the diet, and protein, which has a simila but less marked effect, should be reduced to 30 to 40 grams of casei daily in the form of skimmed milk and skimmed milk powder, an ample supplies of sugar should be given. The dietetic instructio with respect to glucose, protein, and fluid given on p. 301 for acut hepatitis will be found suitable. Fruits, stewed and sieved, a fruit juice may be allowed if desired by the patient. Sulphathiazo or sulphadiazine should be given orally, one of the autho (L. S. P. D.) having shown that these drugs reach the wall of t gall-bladder and also are present in the bile in high concentrati Exercises and drugs to increase the flow of bile and stimulate t gall-bladder to contract are strongly contra-indicated in the acu stage.

When the condition settles down, as it usually does within t or three days, clear soups, beef tea, milk, fruit jellies, and cere may be added until the diet is rapidly built up to that rec mended for chronic cholecystitis (see below).

2. Chronic Cholecystitis, Cholangitis and Cholelithiasi

The treatment of cholecystitis with accompanying cholelithiasi primarily the province of the surgeon. Osler is credited with statement that 'the medicinal treatment of gallstones is a cha in our book of therapeutics the leaves of which are best tur very rapidly'. Nevertheless, the physician has an important to play in deciding on the most suitable time for operation an carrying out pre-operative treatment on the lines described be In addition, the physician must assume responsibility for the tr ment of biliary dyskinesia, non-calculus cholecystitis and calc cholecystitis should this develop in a patient unsuitabl operation because of advanced cardiac or renal disease.

The principles of treatment can be summarized as follows:

1. *Reduction of Weight.* As the majority of these patients are over-weight, the prescription of an anti-obesity diet is of fundamental importance (see Diet No. 24, p. 482).

2. *Improvement of Drainage of the Biliary Tract.* This is needed since stasis is recognized as an important factor in causing and prolonging infections and producing gallstones. It is accomplished by (i) the prescription of special deep breathing and abdominal exercises, and regular exercise in the fresh air; (ii) the use of drugs which stimulate the flow of bile or alter its physico-chemical composition (*e.g.* bile salts), or cause contraction of the gall-bladder, or reduce spasm in the bile ducts or at the sphincter of Oddi (*e.g.* magnesium sulphate, the nitrites, belladonna, and olive oil). Sulphonamides should also be given, for these improve drainage by controlling infection with its consequent catarrh.

3. *Treatment of the Dyspepsia.* This is commonly associated with diseases of the biliary tract and may be of the achlorhydric, hypo- or hyperchlorhydric types (see pp. 276 and 277).

4. *Dietetic Measures*

Fat Content of Diet. We have come to the conclusion that it is both unnecessary and unwise to limit too strictly the fat content of the diet in chronic cholecystitis. The passage of fat into the small intestine is recognized to be not only the physiological but also the most effective means of causing contraction of the gall-bladder and ducts, and thus inducing drainage of the biliary tract. In addition the exclusion of butter, margarine, milk and cream in a chronic disease makes the diet unpalatable and monotonous and low in its content of vitamins A and D. Many textbooks recommend a severe restriction of fat on the grounds that fatty foods are rich in cholesterol, which is an important constituent of gallstones. We disagree with this viewpoint for the reasons stated below. Some restriction of fat, however, is essential if obesity is present or if the patient finds fatty foods cause nausea or indigestion. Natural fatty foods, such as milk and butter, in moderate quantities are in our experience well tolerated, and we allow our patients from ¾ to 1½ oz. of butter or vitaminized margarine (see below) and ½ to 1 pint of milk in the 24 hours, due regard being given to whether obesity is or is not present. Beef and mutton are allowed provided excess of visible fat is removed, as also are white fish, rabbit and chicken; but the fatty meats (duck, goose and pork) and fatty fish (salmon and herring) and fried foods should be avoided because of their indigestibility and because of the tendency to obesity so often present in sufferers from chronic gall-bladder disease.

Restriction of Cholesterol. Many authorities recommend tha foods rich in cholesterol should be entirely excluded from the die of patients suffering from cholecystitis or cholelithiasis. Th advice seems to us to require modification for the following reason

1. Endogenous cholesterol metabolism proceeds even when diet free of cholesterol is given.

2. There is no satisfactory evidence that a moderate intake fatty foods increases the liability to cholecystitis or to the form tion of gallstones, while the value of fat in augmenting drainage the biliary system is well established.

3. The incidence of gall-bladder disease is not increased in cc ditions characterized by a high blood cholesterol (*e.g.* diabet mellitus and lipoid nephrosis).

4. Investigations carried out under the direction of one of t authors (Davidson, 1944) showed that there was no necess correlation between the intake of cholesterol in the food and t level of cholesterol in the plasma or bile. Similar values w obtained whether the patients ingested 300 milligrams or 2, milligrams of cholesterol daily. This work fully substantiated conclusions reached by Gardner in 1929.

(5) The results of cholesterol feeding experiments in animals not necessarily applicable to man. It has been clearly den strated that when large amounts of cholesterol are fed to anir (rats, guinea-pigs, rabbits) the value of cholesterol in the bloo raised and the cholesterol and fat content of the liver is mark increased. Cook (1942) points out that if the amount of cholest given in these experiments in animals is translated into tern cholesterol intake from some common foodstuff for man, th man of 65 kilograms weight would have to take 270 eggs a da equal the amount given to the rat, or 54 eggs a day to equal given to the rabbit. It is obvious, therefore, that it is quit justifiable to advocate low-fat, low-cholesterol diets in the t ment of diseases of the biliary tract in man on the basis of animal experiments.

Accordingly it may be concluded that the severe restricti fatty foods with the object of preventing the formation of stones rests on no sound basis. It seems most unwise to wit from the patient the beneficial effects which have been prov result from the ingestion of fat, because of unproven hypo as to the metabolism of cholesterol. It is not generally realize there exists no correlation between the cholesterol and fat co of different foods and yet this is clearly shown in Table 9.

TABLE 9

Cholesterol and Fat Content of Different Foods

Per 100 grams	Cholesterol milligrams	Fat milligrams	Ratio Fat : Cholesterol
Brains	2,500	15,000	6 : 1
Sweetbread	2,500	15,000	6 : 1
Fish roe	300	2,000	7 : 1
Egg (2 without shell)	600	12,000	20 : 1
Kidney	300	7,500	25 : 1
Liver	150	7,500	50 : 1
Meat (lean)	65	13,000	200 : 1
Milk	12	3,600	300 : 1
Butter	250	80,000	320 : 1

From this Table it may be concluded that brains, sweetbread, fish roes, kidney and liver should be excluded from the diet since their relatively low fat content, as compared with that of butter, cream or cheese, will be of little value in promoting biliary drainage, while their high cholesterol content may possibly be a disadvantage. Vegetables and cereals contain small amounts of sterol, most of which is phytosterol and hence not assimilable. Restriction of these foods will obviously not affect cholesterol metabolism. If it were desired to limit the cholesterol content of the diet to a minimum and at the same time provide the physiological stimulus for gall-bladder contraction, it would be rational to replace butter by olive oil or by margarine made from vegetable oil. A diet containing 1 pint of milk, 2 oz. of butter, and an average helping of fish or meat at two meals in the day will yield about 300 to 400 milligrams of cholesterol. This figure corresponds with the figure given by Burger for an average mixed diet taken by an adult. Cook's paper (1942) should be referred to for an excellent review of cholesterol metabolism and for further information on the sterol contents of tissues and foods.

Eggs and Fried Foods. It is a well-recognized clinical observation that many patients suffering from diseases of the liver, gall-bladder and biliary tract are unable to eat fried foods and eggs because of the consequent flatulent dyspepsia. It is easy to understand why they are unable to tolerate excess of fat or fried foods (see p. 167), but the reason for the intolerance to eggs (other than fried eggs) is far from clear. An average helping (3 oz.) of chicken or lean meat contains approximately 24 grams of protein and 9 grams of fat and may be well tolerated, and yet the patient may complain of flatulent dyspepsia if one egg is eaten, although the content of

protein and fat is only about 5 grams of each. In our experience t
idiosyncrasy in some cases is associated especially with the i
gestion of *fried* eggs, and no symptoms may be caused by light
boiled or poached eggs or eggs present in custard or sponge cak
Hence we see no particular merit in excluding eggs from the di
as a *routine* measure for the prevention or treatment of dyspeps
when greater amounts of fat and protein are being consumed
other articles of food. Moreover, there is sound experimen
evidence from both animals and patients with biliary fistulæ
show that egg yolk exerts a remarkably potent effect on bili
drainage. We have frequently allowed our patients with chro
disease of the gall-bladder to take one or two eggs a day if they
desired, and we have never been satisfied that the clinical cou
of the disease was adversely affected in any way. This is
surprising in view of our investigations into cholesterol metabol
referred to above. On the other hand, if it is believed that
cholesterol intake is of importance in the formation of gallsto
then obviously eggs must be prohibited, because there is as m
cholesterol in one egg as in all the rest of the food in an ave
mixed diet.

Other Dietetic Measures. The protein content of the diet sh
be at least 80 grams per day, as protein has been shown to b
effective stimulator of gall-bladder contraction, and to pla
important part in maintaining the functional integrity of the
(see p. 293). Lean beef and mutton, chicken and white fis
allowed in moderate quantity. If there is any reason to believ
hepatitis exists together with the cholecystitis or cholelithias
is not infrequently the case, half of the protein should be giv
casein in the form of whole milk, skimmed milk or skimmed
powder.

The greatest care should be taken to avoid large meal
indigestible articles of food (see p. 265) because of the eas
which dyspepsia occurs in patients suffering from chronic d
of the gall-bladder.

Lastly, but not least in importance, is the need for
quantities of fluids, which should be taken first thing in the
ing and last thing at night, and between meals when the st
is empty.

The fat intake should vary from 50 to 70 grams and the
hydrate from 100 to 400 grams daily, depending on w
obesity is or is not present. Diets Nos. 23 and 24 (pp. 479
will meet these requirements.

CHAPTER XIV

DISEASES OF THE KIDNEY AND URINARY TRACT

At the present time there is considerable diversity of opinion about the value of diet in the treatment of diseases of the kidney. There are some who believe that diet is the chief therapeutic weapon for all forms of nephritis; others hold not only that there is little evidence that diet alters appreciably the course of progressive renal disease, but that rigid dietary restriction, especially of protein, does harm by lowering the patient's general condition and powers of resistance to infection. These conflicting views are at least partly due to the misconception of the metabolic changes caused by renal disease and partly to a failure to define clearly the aims of dietetic treatment in the different types or stages of nephritis. In the writers' opinion, apart from rest in bed and protection from cold, dietetic measures constitute the most important part of treatment in acute nephritis. In subacute and chronic nephritis with œdema, massive albuminuria and hypoproteinæmia, a properly constructed diet is of both symptomatic and general value. In chronic nephritis without œdema but with failure in renal concentrating power, there is much less satisfactory evidence that dietetic treatment will delay to any considerable degree the nal onset of uræmia. For this reason, and also because of the chronicity of the disease, it is unwise to insist on premature or too rigid dietetic restrictions which may depress the patient mentally and impair his physical strength without appreciably improving his renal function.

Just as the discovery of glycosuria does not necessarily indicate that the patient is suffering from diabetes, so too the discovery of albumin in the urine does not invariably mean that renal disease is present. Before a case of albuminuria is treated as one of renal disease, other causes of albuminuria must be eliminated, especially the functional or orthostatic albuminuria of young persons and the albuminuria of chronic venous congestion in elderly persons suffering from hypertensive heart disease.

Before a patient with albuminuria is subjected to any dietetic restrictions, evidence of impaired renal function must have been obtained. Such information may be obtained in some cases by examining the urinary deposit for red blood cells and tube casts, or from the analysis of the blood for evidence of nitrogen retentio In other cases, special tests of renal function will have to be applie before the significance of the albuminuria can be determined. It beyond the scope of this book to describe such tests as Maclean urea concentration test, or Calvert's modification of this (the ur concentration range), or the Van Slyke urea clearance test, but may be mentioned here that renal function can be tested succes fully by the simple method of assessing the concentrating pow of the kidney from the specific gravity of the urine. Fishbe (1939) has described a method of standardizing the application the test, so that the maximal concentrating power of the kidney brought out and the results of successive tests can be compar The patient receives no food or fluid after 6 p.m. on the even before the test. Before going to bed the bladder is emptied a the urine discarded. On waking next morning, the bladder emptied and the urine preserved (sample 1). At the end of hour the bladder is again emptied (sample 2). The patient gets (if able to do so), and at the end of another hour the bladde emptied a third time (sample 3). The specific gravity of e sample is measured. If the kidney's function is unimpaired, on the samples will have a specific gravity of at least 1,022, and o up to 1,032 in younger patients. Where very severe renal dan is present, the specific gravity of the urine becomes fixed at al 1,010. With lesser degrees of renal damage, the maximum spe gravity of the urine in this concentration test will lie somew between 1,022 and 1,010.

As failure of concentrating power is the earliest sign of ki damage, this test is suitable for the detection of the lesser de of renal impairment. It is only when the failure of concentr power has progressed to a considerable extent that the conce tion of nitrogenous substances in the blood begins to rise.

OBJECTS OF DIETETIC TREATMEN IN NEPHRITIS

If the patient has been shown by any of the above tests suffering from kidney disease, the next question that arises aim of the dietetic measures that are to be employed. The kidney disease should be so arranged as to:

1. *Spare the diseased kidneys* by minimizing their w excreting the end-products of metabolism. In some cases, s in acute nephritis, this aids the kidneys in recovering full effi

2. *Prevent uræmia.* Accumulation of waste products of metabolism leads to uræmia, and this tendency should be combated by reducing the amount of these waste products and by measures to promote their excretion by the damaged kidneys.

3. *Prevent œdema,* which is caused by retention of water and salts.

4. *Maintain nutrition.* It is apt to be forgotten that chronic nephritis is essentially a wasting disease. Therefore the diet should have a caloric intake which is adequate to maintain the patient's nutrition and should contain optimal quantities of protein, vitamins and minerals.

PRINCIPLES OF DIETETIC TREATMENT IN NEPHRITIS

To achieve the objects mentioned in the preceding section, the diet must be modified in some or all of the following important factors:

1. The protein content.
2. The salt content.
3. The acid- or base-forming qualities.
4. The water balance.

1. The Protein Content

It is still an open question whether a high protein diet has a harmful ffect on the kidneys either in causing renal disease or in accelerat ng the progress of the disease once present (see p. 30). On the heory that albuminuria indicated a failure of the body to utilize rotein and that the powers of the kidney to excrete the nitro enous end-products of protein metabolism were damaged, restric ion of protein in the diet used to be carried to extreme lengths, in rder that protein metabolism might thereby be greatly decreased, lbuminuria diminished, and the amount of nitrogen to be excreted educed to a minimum.

It is now known that it is impossible to obtain a protein-free etabolism, even when protein is completely excluded from the iet. The 'wear and tear' of the bodily processes leads to a constant eakdown of a certain amount of body protein – the so-called ndogenous protein metabolism, which cannot be reduced by any ietetic measures and so constitutes an irreducible minimum of rotein metabolism. Endogenous protein metabolism probably volves the breakdown of some 40 grams of body protein daily, though some workers claim to have obtained nitrogenous equilib-

rium on protein intakes as low as 25 grams. Restriction of protei
in the diet below the amount required to make good this constan
breakdown of body protein will not reduce protein metabolism
but will merely force the body to break down its own protein t
supply its needs, so that the patient's health will be furthe
impaired by under-nutrition.

The former practice was to restrict the protein intake in all typ
of nephritis. It is now known that this is irrational and, in man
cases, may actually be harmful by impairing the patient's gener
health. The only justification for reducing the protein intake
chronic nephritis is an impairment of the efficiency of the kidn
in excreting metabolic waste products, as shown by a reducti
in concentrating power. Protein restriction is also indicated
acute nephritis, on the grounds that reduction of the kidney's wo
to a minimum will rest that organ and aid recovery.

The extent to which protein restriction should be carried cann
of course, be judged from the degree of albuminuria. The excreti
of albumin in the urine is an indication of a pathological incre
in the permeability of the walls of the glomerular capillaries wh
normally do not allow the passage of proteins. The urinary prot
is probably derived directly from the plasma proteins, and
differs from the other nitrogenous substances in the urine in tha
is not an end-product of metabolism, but merely protein lost to
body. Albuminuria *per se* is therefore an indication not for pro
restriction but rather for a liberal intake to replace this wast
protein.

The desirable protein intake likewise cannot be judged solel
the presence or absence of nitrogen retention in the blood. R
efficiency is impaired long before evidence of nitrogen retentic
the blood appears, and if restriction of protein in the diet is g
to accomplish anything, it is logical to apply it in the earlier st
of impaired renal function. Therefore, the protein intake shou
regulated by the results of renal efficiency tests, such as the si
concentration test of Fishberg described on page 310. The a
extent of protein restriction advised is discussed when dealing
the different forms of nephritis, but it is a safe general rule th
protein quota should not be less than 0·5 gram per pound of
weight, except in acute or subacute nephritis or in states of
insufficiency with marked nitrogen retention.

These recommendations are based on available knowledge
the effects of high and low protein diet on healthy and di
kidneys and on our clinical experience and nitrogen balance e

ments. We have discussed the first of these problems on p. 30. For further relevant data the reader is referred to a review by Mosenthal (1931). In our opinion there is no satisfactory proof that an intake of 100 to 200 grams of protein daily has any deleterious effects on the healthy kidney. Moreover, there is evidence, both in human and animal experiments, that the change from a low protein to a high protein diet may markedly improve the urea clearance. The effect of the protein intake on a diseased kidney is much more difficult to assess, but the following observations are worthy of consideration.

One of the authors (L. S. P. D.) had a kidney removed for hydronephrosis at the age of 12. Despite this handicap he managed to enlist in the infantry in August 1914 and spent the autumn, winter and spring in the trenches in Northern France under the dreadful conditions of wet and cold that existed during that period of the First World War. In the summer of 1915 he was very severely wounded and developed extensive gas gangrene. His life was seriously endangered from toxæmia, sepsis and cachexia for nearly a year. That his remaining kidney was markedly affected was manifested by the constant passage of albumin, leucocytes and casts in the urine. The diet prescribed and eaten for more than a year after discharge from hospital was low in protein and high in carbohydrate, and alcohol was prohibited. As the albuminuria continued and health was not fully restored, and because the restriction of food and drink proved extremely irksome, he decided to stop all dietary restriction and eat and drink whatever he liked. Nearly thirty years have elapsed since that decision and the function of the one remaining kidney appears to be perfectly satisfactory, as judged by the sense of well-being and the absence of abnormal constituents in the urine and the blood.

The high protein diet used to counteract the hypoproteinæmia in the nephrotic syndrome has never been shown to have any deleterious effect on renal function. On the other hand, low protein diets tend to produce anæmia, lowered vitality and reduced resistance to infection. The potential danger to the liver of a deficient supply of protein is discussed on page 293. This aspect of the matter is of particular interest since in some cases of nephritis the degree of hypoproteinæmia present cannot be accounted for by the amount of albumin lost in the urine, and the possibility must be considered that its cause lies in injury to the liver by retained toxic metabolic products.

Our experience of the benefits which may result from increasing the intake of protein in certain types of nephritis with and without nitrogen retention is in keeping with that of Aldrich and Boyle discussed on p. 327. Thus, we are satisfied that it is most desirable to prescribe sufficient protein to keep the nephritic patient in nitrogenous equilibrium and to prevent the development of anæmia and hypoproteinæmia. Nothing will be gained by exceeding this intake, since to do so merely increases the work of the kidneys and raises the level of nitrogenous waste products in the blood without any corresponding benefit to the patient.

Most authorities state that 40 grams of protein daily will meet these requirements. Carefully controlled nitrogen-balance experiments, carried out by O. Olbrich under the direction of one of the authors (L. S. P. D.), have shown that, while this is true of norma persons, many nephritics with nitrogen retention are in negativ nitrogen balance while in bed on a diet containing 2,000 Calorie and ¾ gram of protein per kilogram body weight – *i.e.* 50 grams fo a man of 70 kilograms (140 lb.). To continue in negative nitroge balance invariably results in progressive loss of weight an strength. Accordingly we advocate that the protein intake shoul not be reduced below 50 to 60 grams daily even when the bloo urea is as high as (or even higher than) 100 mg. per 100 c.c. Whe signs of impending uræmia appear it may be advisable to reduc the intake of protein to 40 grams or even less for a few days whi fluids are being forced to increase diuresis. A return to a diet whic maintains nitrogenous equilibrium must be made, otherwise th patient will die from inanition and malnutrition. When the ren output of urine finally decreases to a level which is not sufficie to clear the blood of products of protein metabolism, the patient end is approaching and it is neither helpful nor kind to prohibit a reasonable demands he may make.

Another fallacy which has been exposed is the old concepti that 'red' meat is more harmful to patients with nephritis th 'white' meat (see p. 176). Actually, red and white meats ha practically the same content of purines and extractives, and wh it is remembered that it is only the end-products of protein met bolism which reach the kidneys, it is obvious that it is of lit importance whether these end-products were originally deriv from the proteins of meat, fish, eggs, or cheese. When prot restriction is necessary, the object of treatment is to maint nitrogenous equilibrium on the reduced protein intake, so that protein quota of the diet should include proteins of the high biological value obtained from meat, fish, or dairy products.

Most clinicians rigorously prohibit the ingestion of alcoh beverages on the grounds that alcohol is a renal irritant. T concept dates back to the time of Bright, but there is little evide that alcohol in moderate quantities causes any harmful effects the kidney. Since alcohol is very largely oxidized to carbon dioxide and water this is not surprising. We calculate that not more than 1 c.c. of alcohol will be excreted by the kidneys over a period of 1 to 2 hours after the ingestion of one small whisky (1 oz.), ¾ pint of beer, or 4 oz. claret. Mosenthal (1931) describes the investigations of Bruger, Locales and Guthrie on the effects of alcohol on the urinary constituents and renal functions of patients suffering from nephritis of various types, and comes to the conclusion that 'alcohol in sensible amounts may be used to stimulate the appetite or increase the caloric value of the food in any form of nephritis except arteriosclerotic nephritis'. Such advice may sound revolutionary, but medical advice should only be given when it is backed by controlled experimental and clinical trial. It would have been equally revolutionary to suggest, even a few years ago, that protein should not be restricted when albuminuria was present. The danger of protein restriction in hepatic disease, now clearly demonstrated, illustrates in another field how erroneous advice based on theoretical considerations can influence clinical practice for long periods. As already mentioned, one of the authors has never restricted his reasonable demands for alcohol, despite the fact that he has only one kidney which was severely damaged in 1915–16, and no harm has resulted. We do not wish to imply that any therapeutic benefit is obtained by prescribing alcohol for nephritis or that alcohol is not a renal irritant if taken in excessive amounts. We merely suggest that before *entirely* prohibiting the use of alcohol to a chronic nephritic who enjoys his *occasional* glass of beer, wine or whisky, and who perhaps has not many years to live, the doctor might do well to carry out some simple observations to test the correctness of Bright's views put forward more than one hundred years go.

2. The Salt Content

It was formerly believed that retention of chloride ions or sodium ions in the body, owing to a failure of the kidney to excrete them, was the cause of œdema in renal disease, water being retained along with the salt to preserve the normal osmotic tension of the body fluids.

While there is no doubt that the degree of œdema in nephritis may be altered materially by varying the intake of salt, the theory that salt retention is the primary cause of the œdema cannot be maintained. It may be recalled that in the complete anuria which sometimes develops in such conditions as nephrolithiasis and cortical necrosis, marked retention of sodium chloride occurs and its concentration in the plasma rises if the patient continues to ingest salt, and yet œdema does not appear.

The œdema of kidney disease may be due to any of the three main factors leading to œdema formation:

1. *Increased Permeability of the Capillary Walls*, so that the proteins of the plasma as well as dissolved salts can pass out into the tissues. This is considered to be the cause of the œdema of acute nephritis, the increased capillary permeability presumably being caused by the same toxic agency as that which damages the glomerular capillaries.

2. *A Fall in the Colloid Osmotic Pressure of the Plasma.* I chronic nephritis with œdema, there is a fall in the level of th plasma proteins (chiefly the albumin fraction), due either to los of protein in the urine or to defective synthesis of the plasm proteins. It is the osmotic pressure of the plasma proteins whic normally counteracts the hydrostatic pressure of the blood in th capillaries, the latter tending to force fluid outwards through th capillary walls. When the plasma protein falls from the norm 6 to 8 grams per 100 c.c. to a level below 5 grams per 100 c.c., th capillary blood pressure is no longer adequately opposed, and flu passes from the capillaries into the tissues.

3. *Increased Blood Pressure within the Capillaries*, which occu in the chronic venous congestion of cardiac failure. The œdem found in chronic nephritis with hypertension is frequently cardi in origin.

While retention of sodium chloride is not the cause of any of t types of œdema seen in renal disease, the ingestion of salt w aggravate the œdema caused by any of the three factors mention above, because, for some reason not fully understood, the sodiu ion facilitates the formation of œdema. The restriction of salt the diet is indicated in nephritis with œdema, but there is justification for indiscriminate reduction in salt intake in all for of nephritis. In chronic nephritis with hypertension but no œder salt restriction is not called for unless cardiac failure superver and it may be actually harmful by producing lack of appetite consequent undernutrition.

A strictly 'salt-free' diet is unattainable. Most foods are low salt, and sufficient salt restriction is usually obtained by prohi ing the use of salt in cooking or at table, and forbidding the us salty foods such as bacon, ham, salt and corned meat, n extracts, sausages, salt butter, cheese and smoked fish and sardi

In acute nephritis and in congestive heart failure, the diets used are low in Calories and protein and rich in carbohydrate, *e.g.* fruit and fruit juice, cereals, sugar and jams – foodstuffs which have an extremely low salt content. Such diets yield about 2 grams or less of sodium chloride daily, a considerable reduction from the 10 to 15 grams which is a normal daily intake. In chronic nephritis with œdema but without failure in renal concentrating power, the diet recommended is essentially a normal one, except for an increase in the intake of protein and a restriction of fluid and salt. Even when salt is not used in cooking or at table and salty foods are prohibited, the sodium chloride content of such a diet will be in the neighbourhood of 2 to 4 grams daily. To bring the salt content to below 2 grams daily, it is necessary to replace bakers' bread with a low salt casein bread, and to use special procedures in the cooking of meat and vegetables with the object of reducing their salt content. It is not possible, except in properly equipped institutions, to carry out such a programme. Fortunately, it is extremely rare for such low salt diets to be required because the œdema can usually be efficiently removed by raising the plasma protein level and by the use of diuretics.

Considerable difficulty may be experienced in persuading patients to keep to a low-salt diet, as such a diet is unappetizing and its long-continued use may lead to anorexia, with the attendant danger of under-nutrition. When the patient's condition deteriorates with its use, a low-salt diet must not be continued. The palatability of a low-salt diet can be improved by the judicious use of other flavouring substances such as lemon juice, vinegar, mustard, pepper or spices. Many 'salt substitutes' have been introduced, but as most of these are salts of sodium, their use is illogical since it is accepted that it is the sodium ion which tends to aggravate œdema formation. The use of potassium chloride is permissible, however, as potassium salts have a diuretic action. 'Seloram' (Bayer) is a suitable salt substitute of this nature.

3. Acid or Base Forming Properties

Besides regulating the osmotic pressure of the body fluids, the kidneys also maintain the optimal pH of these fluids by excreting the acid end-products of metabolism. A diet with an acid ash, therefore, increases the work of the kidneys, and may possibly further impair the efficiency of damaged kidneys and increase the tendency to acidosis in nephritis.

Good results have been claimed for the treatment of nephritis by the use of diets yielding an alkaline ash. Such diets must b rich in milk, fruit and vegetables and poor in meat, fish, eggs bread and other cereals.

The calculation of the acid- or base-forming quality of a diet i a somewhat involved procedure. In addition, a marked restrictio in the acid-forming foods makes the diet unappetizing and monotonous. Accordingly, since many authorities claim that as good better results can be obtained by prescribing alkalis (*e.g.* potassiu citrate 30 to 60 grains t.d.s.) along with an ordinary diet, we fe that the acid- and base-forming properties of the diet have be unduly emphasized in the past. (For a full discussion of th problem see p. 341.)

4. The Water Balance

In nephritis with œdema, the output of urine is decreased. Th may be evidence of impaired renal function, as in acute nephrit or due to the pre-renal deviation of water to œdema fluid by a of the causes mentioned on page 316, so that the amount of wa reaching the kidneys for excretion is reduced. Whichever of th causes is operative, the ingestion of large amounts of water aggravate the œdema. Therefore, restriction of the fluid intak just as important as salt restriction in the treatment of nephr œdema. Where œdema is present, the logical procedure is reduce the fluid intake to a level below that of the fluid lost fr the body, so that the œdema may be reduced. In Chapter VI was shown that the fluid intake from 'solid' food plus the w formed in the oxidation of the food in the body was approxima equal to the water lost from the lungs and skin. Therefore, attempt should be made in œdematous patients to keep the all ance of fluid taken as such below the urinary output. In extr oliguria, such as occurs in many patients with acute nephritis, is impossible, however, and in practice it is found that a intake of below 1 pint cannot be tolerated and even an intak 1 pint daily can seldom be maintained for more than two or t days. Excessive fluid restriction leads to weakness, anorexia irritability. As long as there is œdema, the fluid intake sh however, be restricted to 1½ to 2 pints daily.

Where facilities are available, the fluid balance may be culated. The intake is found by adding 80 per cent of the w of the 'solid' food to the amount of fluid taken as such, whil excretion of water is taken as the urinary output plus 1,20 (water lost from lungs and skin). A simpler method of contr the fluid balance is by the daily weighing of the patient; changes in weight from day to day will represent changes in water content, provided the diet is adequate in Calories.

Rigid fluid restriction in nephritis with œdema should only be practised when the renal function, as judged by the concentrating power of the kidney, is good or shows only moderate impairment. In cases where the concentrating power is markedly diminished, and where there is a possibility of uræmia supervening, as shown by nitrogen retention in the blood, radical fluid restriction is dangerous, as it may precipitate uræmia. The kidney can only excrete waste products in dilute solution and, therefore, if the intake of fluid is much reduced, insufficient water may reach the kidney to secure adequate elimination of waste products. In such cases, although œdema may persist and even increase, a more liberal fluid intake must be allowed.

In nephritis without œdema, there is no indication for fluid restriction and the fluid intake may be left to the patient's own desire.

DIET IN DIFFERENT TYPES OF NEPHRITIS

The aims of dietetic treatment are not identical in the different types or stages of nephritis, so that it is essential to have a simple working classification of the different forms in which nephritis may be encountered. Since dietetic treatment in renal disease is largely symptomatic, and is regulated by the degree of renal damage rather than by the precise pathological condition producing this damage, the differentiation of the various clinical types of nephritis will be based largely on the predominant symptoms and the state of renal function. The following classification of nephritis will be used in discussing the dietary treatment of renal disease:

A. Acute nephritis.

1. *Acute diffuse (glomerulo-tubular) nephritis.*
2. *Acute focal nephritis.*

B. Subacute and chronic nephritis with albuminuria and œdema.

1. *Without hypertension and without failure in excretion of nitrogenous waste products (the nephrotic syndrome).*
2. *With hypertension and with failure in excretion of nitrogenous waste products.*

C. Chronic nephritis without œdema but with hypertension and failure in excretion of nitrogenous wast products.

1. *Terminal or third stage of B 2 (chronic nephritis).*
2. *Terminal stage of essential hypertension and nephrosclerosis.*

It must be admitted that it may be difficult to classify som cases of chronic nephritis into one or other of the above categorie This is only to be expected if it is remembered that the diseas process is a progressive change passing from acute nephriti through stages B 1 and B 2 and finally terminating in stage C 1.

Nephrosclerosis is placed in group C with chronic nephritis in th terminal azotæmic phase because the resulting renal failur although caused by a different mechanism, requires the san therapeutic measures. The chronic renal lesion which leads death from uræmia in under 10 per cent of cases of essential hype tension is not due to an inflammation of the kidney and hen should not be called 'nephritis'. The pathological change is fibrosis secondary to ischæmia which slowly destroys the nephro

During the transition of nephritis from the subacute to t terminal chronic stage, albuminuria lessens in degree and may ev disappear, while œdema diminishes. There are several explanati for these changes, but the one which seems to be most accepta is that progressive fibrosis leads to a cutting off of the blood sup to the damaged glomeruli and tubules. In consequence, the leak glomerular filters gradually disappear, with a concomitant red tion in the loss of albumin in the urine. With the reduction albuminuria the level of the plasma protein rises. Another ef of the fibrosis and destruction of nephrons is a reduction in power of the kidney to concentrate nitrogenous waste produ To maintain the excretion of these products the kidney secr large amounts of dilute urine. The combination of polyuria the increase of the plasma protein explains the progressive appearance of the œdema. Concomitantly there appears evidence of impairment of renal function, *e.g.* urine of low and f specific gravity and a rise in the blood urea.

A. Acute Nephritis

1. *Acute Diffuse Nephritis*

This disease occurs most commonly in children and young ad the onset frequently occurring one to three weeks after a str coccal infection, usually tonsillitis or scarlet fever. The generalized toxic damage to the glomeruli and, to a lesser extent, to the tubules. Characteristically, there is albuminuria, hæmaturia and oliguria, epithelial and granular casts in the urine, œdema of varying degree and hypertension. Mild forms of the disease may show little general upset, and the œdema and hypertension may be transient or even absent; in severe cases, the patient complains of headache, nausea and vomiting, hypertension and œdema are marked and there is considerable nitrogen retention in the blood.

The important principles in the dietetic treatment of acute nephritis are:

1. *A marked reduction of the protein intake.* On the basis of resting an inflamed organ, the aim is to reduce the protein metabolism to the lowest possible level (*i.e.* the level representing endogenous protein metabolism), and so lessen the work of the kidney by reducing the amount of the end-products of protein metabolism to be excreted.

2. *A low salt intake.* In acute nephritis there is a generalized increase in capillary permeability, and hence œdema. It is therefore rational to reduce the salt intake so that the œdema should not be aggravated.

3. *Restriction of the fluid intake.* This is also necessary to combat he formation of œdema fluid.

4. *A low Calorie intake.* This reduces metabolism and therefore ends to lower the circulation rate and the blood pressure, which may help to lessen the formation of œdema. Acute nephritis is a isease of comparatively short duration, and brief starvation does no harm.

The treatment in severe and mild cases may conveniently be onsidered separately.

1. *Severe Cases.* It is a widely accepted custom to begin reatment with a period of absolute protein restriction, only ruit juice and sugar being given. Such treatment is held to inimize the burden on the kidney by giving it no exogenous itrogen to excrete. It was pointed out earlier, however, that dogenous protein metabolism cannot be reduced by stopping intake of protein, and there is no difference between the endroducts of endogenous and exogenous protein metabolism. On heoretical grounds, therefore, nothing is gained by reducing he protein intake below 30 to 40 grams daily, and, in fact, loss the patient's own body protein will follow reduction below this vel.

From the practical point of view, however, the restriction of t diet in the initial stages to fruit juice is justified in cases wi marked nausea and vomiting, for this type of diet is usually t only one the patients will tolerate. For such patients, 1 pint orange juice diluted with ½ pint of water and sweetened to tas should be given daily in small two-hourly feeds. Alternatively 30 per cent solution of glucose in water flavoured with the jui of an orange or a lemon, or the fruit juice and barley water reci given on p. 419 may be used. Besides being better tolerated some patients than fruit juice alone, it has the advantage o larger Calorie content, 2 pints supplying about half the ba energy requirements. An alternative almost protein-free diet Diet No. 25, given on p. 484, which contains a certain amount solid food in the form of fruit served in a variety of ways. T total fluid content of the diet is about 1½ pints, and ½ pint water may be given in addition. It may be used from the st if patients will tolerate it, or it may be used after the patient had nothing but fruit juice for a day or two. Some authorities (Cameron, 1939) have recommended that 1 pint of fruit juice 1 pint of milk should be given daily from the start, since they h found that no increase in nitrogen excretion results from ingestion of the 20 grams of protein contained in 1 pint of m Since it cannot be said that the relative merits of the protein- fruit or fruit juice diets and the milk and fruit juice diet are fi settled, the practitioner is at liberty to adopt whichever die prefers.

The fluid intake, as will be seen, should be restricted to 1½ pints daily, depending on the degree of oliguria. This is suffi to secure adequate drainage, for the presence of œdema indi that there is plenty of fluid in the body for the kidney to ex as soon as its function improves.

Except in rare cases which progress rapidly to renal failu spite of any treatment, the blood pressure falls, diuresis oc and the œdema disappears after a few days. When this oc the patient should be placed on a diet more nearly approxim to his basal protein and Calorie requirements. Even if there apparent improvement, it is unwise to continue the protei diet or the milk and fruit juice diet for more than seven to ten The next diet should therefore contain approximately 40 gra protein and about 1,800 Calories. The protein quota is d from dairy products (milk and eggs) and the proteins of the c and vegetables. Almost 50 per cent of the protein of Diet

(p. 484) is of high biological value, so that nitrogenous equilibrium should be assured. Meat is omitted, not because of any supposedly deleterious effect of its proteins, but merely to keep the protein intake low.

Salt should not be used in cooking, and none should be added after the food comes to the table. The intake of fluids as such should still be restricted to 1½ to 2 pints.

Further additions to the diet depend on improvement in the kidney function, as judged by renal function tests applied at weekly intervals, the concentration test described on page 310 being the easiest and most satisfactory for repeated use. When the specific gravity reaches 1,018 the protein intake should be increased to about 60 grams by the addition of a small serving of meat, chicken or fish (2 oz.) at one of the meals (Diet sheet No. 27, p. 486). If, however, after two or three weeks on the 40 grams protein diet (No. 26, p. 484), no improvement in renal function occurs and particularly if the patient is losing weight and strength, it must be assumed that nitrogenous equilibrium on 40 grams protein daily is not being secured, in which case the protein intake must be increased to 60 grams, as indicated above. Progress towards restoration of normal kidney function, as shown by the specific gravity of the urine in the concentration test reaching or exceeding 1,020, is an indication for a further increase in the protein quota to an essentially normal level (80 to 100 grams), secured by the addition of another small serving of meat or fish, or by an increased intake of milk, eggs or cheese.

It is now believed that these more generous protein allowances help rather than hinder the restoration of normal renal function, and they certainly aid the recovery of the general condition, which deteriorates with very restricted protein intakes. It should be noted that the persistence of albuminuria is not an indication for modifying the dietetic measures outlined above, provided that the concentrating power of the kidney is not markedly impaired. As mentioned earlier, albumin in the urine represents protein lost to the body, and is an indication for a generous rather than a restricted protein intake to make good this wastage.

2. *Mild Cases*, with little general disturbance, hypertension, edema or nitrogen retention in the blood, do not require the initial 'starvation' period, as they can tolerate a diet supplying their minimal protein requirements from the start. Such patients should at once be given Diet No. 26 (p. 484), and the further additions to the diet should then be made according to the results

of renal function tests, as described when dealing with the treatment of severe cases. When anæmia is present, iron in full doses is of the greatest value.

2. *Acute Focal Nephritis*

This condition may occur in the course of various infections, chiefly those of streptococcal origin, and is most frequent in tonsillitis. It usually develops at the height of the infection, and is characterized by the appearance of hæmaturia, albuminuria and the presence of granular and blood casts in the urinary deposit. There is no œdema or hypertension, however, and renal function is unimpaired. Pathologically, there are small focal inflammatory lesions in the glomeruli, and, to a lesser extent, in the tubules and interstitial tissue. These lesions are evidently due to the actual presence of the causative organism, which can be demonstrated in the lesions and also in cultures made from the urine.

It is important to distinguish focal from diffuse nephritis, for the former causes no impairment of renal function and clears up completely, usually within a few days, having no tendency to become chronic. The absence of œdema and hypertension is the most important point in the differential diagnosis. Focal nephritis is merely an incident in the causal infection, and has no effect on the prognosis of the latter, although the renal complication is more apt to occur in severe infections.

The treatment of focal nephritis is that of the primary infection. The renal complication does not require the dietetic measures used in acute diffuse nephritis to be so strict or so prolonged. It is usual to give a low protein diet until the hæmaturia and albuminuria have subsided, and the diet containing 44 grams of protein given in Diet No. 26 (p. 484) is a suitable one for such a case. No fluid restriction is necessary. Within a week or two the hæmaturia and albuminuria will have disappeared, and a normal diet can be resumed.

B. Subacute and Chronic Nephritis with Albuminuria and Œdema

1. *Without hypertension and without failure in excretion of nitrogenous waste products (the nephrotic syndrome)*

This condition may follow directly upon an attack of acute diffus nephritis, and should be regarded as the second stage of glomerulonephritis; in many cases, it develops insidiously, with no history o a previous acute attack.

A small group of cases, mostly children and young adults, is often classed as a separate clinical entity – *lipoid nephrosis*. In this condition the pathological process is confined to the renal tubules, and there is said to be no tendency to the development of hypertension and renal failure. It is still an open question, however, whether this condition is not merely a variety of chronic nephritis with œdema.

The nephrotic syndrome is characterized by:

(i) Marked œdema, oliguria and a heavy albuminuria.
(ii) Decrease in the plasma proteins, especially in the albumin fraction.
(iii) Increase in the blood cholesterol.
(iv) Absence of cardio-vascular changes
(v) Absence of impaired renal function and nitrogen retention
} In the early stages at least.

The salient clinical features in this syndrome are the marked œdema, the heavy albuminuria and the low level of the plasma proteins. Treatment is therefore directed chiefly towards relief of the œdema and the maintenance of the patient's general condition. The latter is very important, as patients suffering from this disease show an increased susceptibility to infection.

The diet should therefore meet the following requirements:

(i) *Adequate protein intake.* This is permissible, as nitrogen excretion is unimpaired. It is necessary for the maintenance of the patient's general condition and for the replacement of the plasma proteins lost in the urine. The protein intake should be essentially normal – about 100 grams or even more.

(ii) *Restricted fluid intake.* This, along with salt restriction, is the chief method of combating the marked tendency to œdema formation resulting from the fall in the plasma proteins. The daily intake of fluids as such should be restricted to 2 pints or less daily.

(iii) *Low salt content.* It is advisable to reduce the salt intake by prohibiting the use of salt both in cooking and at table, and by forbidding salty foods (see p. 317).

For a case of chronic nephritis with œdema but without failure in renal concentrating power Diet No. 28 (p. 488) will be found to be suitable; it contains about 100 grams of protein. Additional carbohydrate food can be given if desired.

In addition to these dietetic measures for relieving œdema, the use of diuretics must be mentioned, although these are not likely to prove of much value unless the concentration of the plasma proteins can be raised. Urea may be given, since renal function is unimpaired, the dosage being 10 to 15 grams thrice daily. The organic mercurial diuretics may be employed if there is no evidence of acute renal damage, but the urine must be watched carefully for the appearance of blood and casts, and the use of the drug stopped at the first sign of these. Mercurial poisoning has been known to follow the use of these drugs in chronic nephritis.

If the œdema is not relieved by the dietetic and diuretic measures outlined above, it is justifiable to try the effect of a high protein diet such as Epstein recommends in nephrosis. The rationale of this diet is an attempt to make good the marked loss of protein in the urine and so raise the plasma protein level to normal. Such treatment is perfectly safe when the renal function has been proved to be normal, as the kidney can easily excrete the end-products of the increased protein metabolism. Protein intakes as high as 250 grams have been suggested, but patients cannot tolerate such large quantities of protein in the diet day after day, and a moderately increased protein quota of about 150 grams daily is preferable. Since the plasma lipoids are elevated in nephrosis, most authorities recommend that the diet should also be as low in fat as possible. Fluid and salt restriction should be practised as before, but a little salt may have to be used in cooking to render the large helpings of meat and fish palatable. Diet No. 29 (p. 490) is suitable for this purpose. It contains about 150 grams of protein, and a low fat quota (70 grams). Good results often follow the institution of this treatment, although the concentration of the plasma protein is seldom raised to normal, the successful result being attributed by some workers to the diuretic action of the urea which is formed in large amounts in the increased metabolism of protein. If there are no signs of improvement in about three weeks, it is doubtful whether it is of any use persisting with this type of diet.

2. *With hypertension and with failure in excretion of nitrogenous waste products*

In this type of chronic nephritis, marked œdema and massive albuminuria are still the salient clinical features, but there is hypertension and renal function is impaired. The blood shows a low plasma protein level. There is no nitrogen retention in the blood at first, but as the degree of impairment of renal function increases the quantity of non-protein nitrogenous substances in the blood rises. This is one of the most serious types of nephritis, as the downhill course towards uræmia is usually relatively rapid.

The dietetic measures should again be directed mainly towards relief of the œdema, but the protein intake will have to be modified according to the degree of kidney damage as revealed by renal function tests. The dietetic measures to relieve œdema are, of course, a low salt diet (*i.e.* no salt used in cooking or at table and no salty foods given) and the restriction of the fluid intake to 1 to 1½ pints daily. This rigid fluid restriction is only permissible when the kidney damage is not severe. When the concentrating power of the kidney is definitely impaired (*i.e.* the maximum specific gravity of the urine below 1,018) and there is evidence of nitrogen retention in the blood, indicating the possibility of uræmia, the fluid intake must not be reduced below 2 pints. Rigid fluid restriction at this stage may precipitate uræmia, as was mentioned in dealing with the water balance in nephritis.

The protein intake is likewise adjusted to the state of the kidney function. When the maximal specific gravity of the urine is about 1,018 and the blood urea shows little or no increase, a normal diet containing approximately 70 to 80 grams of protein should be allowed. This supplies an adequate reserve to meet the loss of protein in the urine and will help to counteract the tendency to hypoproteinæmia and anæmia which is commonly present even at this stage of the disease. When the maximal specific gravity falls below 1,015 or the blood urea shows a considerable increase, further restriction of protein is necessary. It is absolutely essential, however, to keep the patient in nitrogen balance. The former practice of reducing the intake of protein to 30 to 40 grams has been shown to result in malnutrition, anæmia or lowered resistance. The careful observations of Aldrich and Boyle (1933) on a group of children suffering from chronic nephritis with œdema, hypertension and nitrogen retention clearly indicated the benefits which accrued when their diet was changed from one low in protein to one in which the protein intake was adequate. Not only was the general health of the children greatly improved, but no consistent deleterious effects were produced on the hypertension, albuminuria or nitrogen retention. Controlled investigations carried out by O. Olbrich under the senior author's direction provided explanation of these clinical observations. These showed that patients with chronic nephritis with nitrogen retention were unable to maintain themselves in nitrogen balance on an intake of protein (¾ gram per kilogram body weight) which was satisfactory in this respect for normal individuals. Accordingly we advocate the giving of 50 to 60 grams of protein daily, even when a marked impairment of renal function exists. This advice is tendered not only for the reasons given above, but also because the comfort of the patient must not be sacrificed to an excessively rigid dietary regime when he is suffering from a disease in which the course is downhill and which is invariably fatal. Diet No. 27 (p. 486), already discussed under the treatment of acute nephritis, will be found suitable. Impending uræmia may sometimes be warded off for a time by the use of a protein-free diet (see Diet No. 25, p. 484) for several days, combined with alkalis and a liberal fluid intake to secure the greatest elimination of waste products of which the kidneys are capable. A return to Diets Nos. 26 and 27 must then be made; otherwise the patient will rapidly waste from loss of his own body proteins.

C. Chronic Nephritis without Œdema but with Hypertension and Failure in Excretion of Nitrogenous Waste Products

In discussing the classification of kidney diseases from the point of view of dietetic treatment, it was mentioned that this group included:

1. *Cases in the third stage of glomerulo-nephritis*, in which an attack of acute nephritis has been followed by a period of heavy albuminuria with or without œdema, to be succeeded by the final stage of hypertension, nitrogen retention and polyuria.
2. *Cases of essential hypertension* in which the renal ischæmia and sclerosis are extensive enough to cause impairment of renal function.

The clinical picture is indistinguishable in the two conditions, and as the dietetic treatment is similar in both, no attempt at differentiation is necessary. Actually, chronic nephritis without œdema and of moderate severity has no predominant symptoms, and may only be revealed when albuminuria is discovered during physical examination for some unrelated condition. The only other physical sign constantly present is an elevated blood pressure.

The disease is usually one of long duration, and the rigid dietary restriction formerly enforced probably harmed more patients than it helped, by interfering with nutrition and so affecting the general condition adversely. It is true that by reducing the protein intake to 40 grams or less daily a reduction of the blood urea may occur, but renal function tests show that the state of the kidneys does not improve. More attention is now directed towards the patient's general condition, and the diet is arranged so as to maintain this and, at the same time, relieve the kidneys of extra work as far as possible.

The diet in chronic nephritis without œdema is essentially a normal one, except for the protein content which must be modified according to the degree of impairment of renal function. When the damage to the kidney is only slight, as indicated by a normal blood chemistry but a reduction in the maximal specific gravity of the urine to about 1,018, the patient should be allowed about 70 to 80 grams of protein daily. In practice this means an ordinary diet; the only instruction which need be given to the patient is that he should limit his intake of meat or fish to a *small* helping at lunch and dinner. Ample fruit and vegetables should be taken to maintain an adequate supply of vitamins and minerals. The intake of milk should be about half a pint daily. When the maximum specific gravity of the urine falls to about 1,016 and an increase in the blood urea is found, a further curtailment to about 60 grams is necessary. Diet No. 27 (p. 486) will meet this requirement. It is strongly recommended that the intake of protein should be maintained at this level as long as possible. Severe renal damage, as shown by a maximum specific gravity of about 1,014 or less and a high blood urea, is widely accepted by many authorities as an indication to reduce the protein intake to 40 grams or less daily. The inadvisability of prescribing such low protein diets to nephritics, except for short periods, has been fully discussed on p. 327; but if our advice is not found acceptable, the patient can be given Diet No. 26, p. 484.

As there is no œdema, no stress need be laid on rigid salt restriction, but the patient should be told to be sparing in his use of salt. Restriction of the fluid intake is strongly contra-indicated. The concentrating power of the kidney is reduced so that more water must be excreted in getting rid of the waste products. Fluid restriction would, therefore, tend to aggravate nitrogen retention and precipitate uræmia.

The long duration of the disease makes it essential that any dietary regime must be as little irksome as possible to the patient, whose general condition and sense of well-being are the main criteria by which the effects of treatment are to be judged. Rest, sedatives and reorganization of the patient's activities are probably of greater importance than dietary treatment. It is usual to forbid the use of alcohol unless this imposes a definite hardship on the patient, and tea and coffee should be taken very weak.

When obesity is associated with chronic nephritis of this type, an effort should be made to reduce the weight gradually by the use of a diet moderately restricted in Calories (*i.e.* 1,200 to 1,500 Calories).

Œdema occurring at this stage of nephritis is usually cardiac in origin, and should be treated accordingly, with the proviso that rigid fluid restriction must be avoided.

In all forms of chronic nephritis anæmia tends to develop, and treatment with large doses of iron may lead to considerable improvement in the patient's general condition.

DIET IN SURGICAL DISEASES OF THE URINARY TRACT

Patients about to be operated on for some surgical condition of the urinary tract which has caused impaired renal function and resultant nitrogen retention (*e.g.* prostatic hypertrophy, urethral stricture) should be given a diet rich in carbohydrate and with the amount of protein necessary to maintain nitrogen equilibrium, namely about 50 to 60 grams (see Diet No. 27, p. 486). At least half of the protein should be milk protein. After operation, this diet should be continued until renal function tests show recovery from the kidney damage, when the protein intake can be increased progressively to the normal level (80 grams).

DIET IN INFECTIONS OF THE URINARY TRACT

In contrast to nephritis, in which regulation of the diet forms the mainstay of treatment, infections of the urinary tract must be dealt with by drugs, lavage, etc., and dietetic treatment is of subsidiary importance. Nevertheless, there are one or two points in which attention to the diet may aid the other measures employed.

Acute Cystitis and Pyelitis

The infection is combated in the acute stages by promoting adequate drainage through an increase in the fluid intake, by changing the reaction of the urine so that it is unfavourable to the growth of the infecting organism, and by the use of suitable urinary antiseptics.

During the febrile stage of the illness, a light invalid diet is given, for example the fluid diet set out in Diet No. 35 (p. 498).

In order to increase the urinary output so that the urinary passages may be flushed out mechanically, the patient must increase his intake of fluids to at least 5 pints daily in the form of water, barley water, fruit drinks, and weak tea. The organisms most commonly found in acute cystitis or pyelitis belong to the coliform group of bacilli, which flourish in an acid urine, and the reaction of the urine should be made alkaline by the use of the citrate, acetate or bicarbonate of sodium.

Urinary antiseptics are more usually employed in the subacute and chronic urinary infections, but the sulphonamide group of drugs is often employed along with this alkaline diuretic treatment. It is often recommended that when sulphonamides are given the ingestion of foods rich in sulphur should be reduced. Eggs and onions are generally prohibited, and the intake of meat, fish and cheese is restricted, for most of the sulphur of the diet is derived from the amino-acids, cystine and methionine, which are present in the proteins of these foods in relatively high concentration. Recent work, however, suggests that these restrictions are unnecessary. The avoidance of purgatives, on the other hand, is of fundamental importance, for when the fluid contents of the small intestine are hurried into the colon decomposition takes place with the production of sulphides. Preparations of sulphur (*e.g.* confection of sulphur) and certain drugs (*e.g.* phenacetin) which, if given in the course of sulphonamide treatment, are liable to produce sulphæmoglobinæmia, must on no account be prescribed. Aspirin, however, appears to be harmless.

Chronic Cystitis and Pyelitis

Every case of pyuria which fails to clear up completely with the measures described above requires a careful medical and urological examination. Genito-urinary tuberculosis, stone, or obstructive lesions, acquired or congenital, may be present and will require appropriate treatment before the control of pyuria can be accomplished. Anæmia, sepsis, constipation and physical and mental exhaustion should be corrected, while the patient's general health should be built up with a well-balanced diet. Plenty of fluid should be drunk. The only dietary restriction necessary is the prohibition of foods considered to be renal irritants, such as spices and condiments.

If urinary antiseptics are being used, it is customary to restrict the fluid intake so that the drugs may be more concentrated in the urine. It is probably unwise to reduce the amount below 3 pints daily if sulphonamide drugs are used. In many cases in which the causal condition cannot be discovered or is irremediable, sma doses of sulphadiazine or sulphathiazole, *e.g.* 1 gram daily, may b taken over long periods without harm to the patient and wit beneficial bacteriostatic effects on the urine.

The Ketogenic Diet

Some years ago it was shown that β-hydroxybutyric acid, one o the ketone bodies, has a bacteriostatic action, and the ketogeni diet (*i.e.* a diet in which the fatty acid : glucose ratio is deliberatel raised above 2 so that ketosis develops) was introduced in th treatment of chronic urinary infections. Although numerou successes were reported, the excessive amounts of butter, crean and olive oil in the diet made it nauseating to many patients.

Fortunately, it was subsequently found that mandelic acid, compound with a composition somewhat similar to β-hydroxybutyric acid but not oxidized in the body, has a similar bacteriostatic action, and its use has superseded that of the ketogenic diet Mandelic acid is given as the calcium, ammonium, or sodium salt During its administration the patient may take an ordinary diet In order that mandelic acid should develop its antibacterial action the reaction of the urine must be distinctly acid (pH 5·3 or less) This is secured by giving acidifying salts, either ammoniun chloride or acid sodium phosphate, in the required amounts. I may prove difficult in some patients to secure the correct acic reaction of the urine, and in these cases the prescription of a die with an acid ash may be helpful. Such a diet should contair cereals and meat in abundance, while the intake of fruit, vegetables and milk is reduced.

The recent introduction of sulphonamide therapy has largely superseded the use of mandelic acid owing to its simplicity and to the fact that it is effective in an alkaline urine.

DIET IN URINARY CALCULUS

The treatment of a urinary calculus, once formed, is surgical, but in patients who have had a calculus removed or who pass 'gravel' medical measures may usefully be adopted. By far the most important is that the patient should drink large quantities of fluid – 4 to 5 pints daily, either as water, barley water, fruit drinks or one of the mineral waters.

When the composition of the calculus is known, further measures can be adopted, designed to limit the intake of foodstuffs containing the salt which crystallized out to form the stone. The most common types of renal stone are the urate, phosphate and oxalate stones.

When uric acid tends to precipitate out in the urine, its excretion should be reduced by forbidding foodstuffs rich in nucleo-proteins, *e.g.* liver, kidney, sweetbreads, etc. (see p. 336), and purine-rich beverages such as tea, coffee and cocoa, and by reducing the protein intake. A diet similar to that given for use in chronic gout (Diet No. 31, p. 493) is suitable. Moderate doses of alkalis should be given to render the urine just alkaline to litmus.

Phosphatic calculi are formed in an alkaline urine, and where there is known to be a tendency for the formation of such stones, the urine should be kept acid with small doses of ammonium chloride. Owing to the wide distribution of phosphorus in the foodstuffs, it is difficult to reduce its intake significantly.

The presence of a persistently alkaline urine strongly suggests a focus of infection in the urinary tract which demands treatment.

Oxalates may form calculi, or the excretion of oxalate crystals in the urine may cause dysuria and hæmaturia. Like uric acid, oxalates precipitate most readily in an acid urine, so that the reaction of the urine should be kept just alkaline. The intake of foods rich in oxalates should be avoided – these are chiefly fruits and vegetables such as strawberries, rhubarb, tomatoes and spinach. Chocolate and cocoa should be prohibited and tea taken weak and only in moderate amounts. Patients with a tendency to oxaluria should take plenty of milk, since the calcium of milk will combine with oxalic acid to produce calcium oxalate which is not absorbed from the bowel.

Many authorities recommend alkali or acid ash diets as a means of altering the reaction of the urine and hence reducing the liability to stone formation. The authors' views on this subject will be found on pp. 341 and 342.

CHAPTER XV

GOUT AND RHEUMATISM

GOUT

Gout is characterized clinically by acute or chronic attacks of pain, swelling and inflammation of one or more joints, and by the deposition of sodium biurate in the articular cartilages, in tendon sheaths and bursæ, and in the cartilages of the ears.

Although the fundamental cause of the disease has not yet been elucidated, it is known that in gout there is a disorder of purine metabolism, probably hereditary, the most obvious result of which is an abnormally high level of uric acid in the blood.

Uric acid is the end-product in man of the metabolism of the nucleo-proteins, which are constituents of the nuclei of cells. Actually, only about 50 per cent of ingested purine is excreted as uric acid in the urine; the remainder is presumably destroyed, but the final product of this destruction is still unidentified, unless it be urea.

Like protein metabolism, the metabolism of the purines can be divided into endogenous and exogenous moieties.

Endogenous Uric Acid

On a purine-free diet, there is a continued excretion of uric acid in the urine, which is fairly constant in amount for the individual. The uric acid is most likely derived from the breakdown of nucleo-proteins of the body tissues. It is frequently stated that endogenous uric acid excretion is greatly increased by muscular exercise, but this is only true if the energy requirements are not fully covered by the caloric intake, preferably in the form of carbohydrate. It can be reduced slightly by drastic reduction of the protein intake.

Exogenous Uric Acid

This comprises the uric acid formed from the purines of the food, and can be reduced at will by curtailing the intake of purine. An increased intake of protein and carbohydrate appears to accelerate uric acid excretion (Folin *et al.*, 1924), while a high fat diet appears to cause uric acid retention (Harding *et al.*, 1927).

Purine Metabolism in Gout

Both endogenous and exogenous uric acid excretion are affected. Between attacks, there is usually a decreased endogenous uric acid excretion, but acute attacks are often marked by great variations in the endogenous output. Usually, this decreases for a day or two before the attack, rising rapidly above the normal at the onset of the attack, and then falling, sometimes to a level lower than that obtaining before the attack commenced. On a fixed intake of purine, exogenous uric acid excretion in a gouty subject is only about half that of a normal. The fact that the increase in the blood uric acid in chronic gout is only moderate suggests that the ability of the gouty patient to destroy purines is not defective. The blood uric acid in gout is above the normal level of 2 to 3·5 mg. per cent. In the early stages of the disease, however, it may be normal between attacks. There is, however, no constant relationship between the serum uric acid concentration and the frequency of attacks or the severity of the clinical features. The combination of an increased blood uric acid and a decreased excretion of uric acid in the urine suggests that the increase in the blood is due to a defective excretion of uric acid by the kidney rather than to any increase in its formation. Attacks of gout may be precipitated by dietary indiscretion, infection or unwonted exercise, but often there is no apparent exciting cause.

Diet in Gout

In the treatment of gout, dietary measures are of the greatest importance. In most patients, a well-planned diet, along with the administration of drugs as required, will considerably lessen the frequency and severity of the attacks.

From what has been said already concerning the excretion of uric acid, it will be seen that the diet in gout should be planned on the following general lines:

1. *The purine intake should be reduced to a minimum.* A purine-free diet is, strictly speaking, unattainable as most of even the so-called purine-free foods contain traces of purine. A very marked reduction of the purine intake can, however, be obtained by prohibiting or greatly restricting the use of foods rich in purines. Table 10 based on the figures published by Abrahams and Widdowson (1937) indicates the purine content of different foodstuffs in descending order of richness.

TABLE 10

Approximate Purine Content of Foodstuffs

Foods rich in Purines	Purine Nitrogen in gram per 100 grams of Food
Herring Roe	0·5 to 0·25
Sweetbreads	
Sardines and other small fish	
Herring	0·2 to 0·1
Liver	
Kidney	
Cod roe	
Veal	0·1 to 0·06
Mutton	
White fish	
Salmon	
Chicken	
Pork	
Beef	
Ham	

Meat extracts and meat soups are rich in purines.

Foods with low Purine Content (*i.e.* less than 0·05 gram of purine nitrogen per 100 grams of food).

Tripe
Legumes
Nuts
Cereals, including bread
Root and green vegetables

Foods free from Purines

Milk
Butter
Cream
Cheese
Eggs
Fruit
Jam
Honey
Sugar

Tea, coffee and cocoa contain the methyl purines caffeine and theobromine. It is still not definitely established whether or not these methyl purines can be converted into uric acid in the body. The urinary uric acid is increased by the administration of caffeine but not of theobromine. It is probably best to allow these beverages to be taken only in limited amounts and very weak. Certain proprietary beverages (Kaffee Hag) from which the purine has been removed can be given more freely.

2. *The total caloric intake should be low.* This tends to reduce metabolism and therefore decrease endogenous uric acid excretion. The quantity of carbohydrate should not be reduced, however, to a level which encourages the breakdown of tissue protein.

Many gouty subjects are obese and plethoric, and in these every effort should be made to lower the body weight to a normal level by the use of a low Calorie, low purine diet.

3. *The diet should be relatively high in carbohydrate and low in fat.* According to laboratory investigations, this should increase uric acid excretion.

4. *Alcohol should be prohibited.* The heavier alcoholic liquors definitely increase the tendency to gout, and wines such as burgundy and port should not be taken by a gouty subject. Beers and stouts, too, have a high purine content derived from the yeast and must be avoided. Spirits are not without harmful effects and in general should be prohibited. For elderly patients accustomed to spirits, in whom the gouty diathesis is mild, small amounts of whisky or gin, well diluted with water, may be allowed. Some gouty patients, but not all, can tolerate light wines such as claret or moselle, and can be permitted to take these in small amounts.

5. *Large amounts of fluid should be drunk*, especially between meals, before breakfast, and last thing at night.

Diet during an Acute Attack

A strict lacto-vegetarian diet low in purine, fat and Calories, and relatively high in carbohydrate should be prescribed during the acute attack and for some days thereafter. Diet No. 30 (p. 491) will be found suitable. Fluids should be taken freely, 4 to 5 pints being drunk daily.

Diet between Attacks and in Chronic Gout

Our ignorance of the ætiology of gout, the variability in the duration of freedom from attacks in different persons and in the same person from time to time, and the absence of any satisfactory yardstick, including estimation of the serum uric concentration, by which the value of preventive measures can be ascertained, are factors which make it extremely difficult to give sound advice on treatment between attacks. Generations of physicians in Great Britain have laid emphasis on the value of dietetic treatment. Recent metabolic and experimental studies in America carried out by Bauer and Klemperer (1944) throw considerable doubt on these claims which are based mainly on clinical impressions. These writers state that from a careful review of the literature they are unable to find satisfactory evidence that dietary treatment influences the clinical course of gout. They were unable to provoke or prevent attacks by giving or withholding food rich purine or alcohol. We do not feel justified at the prese entirely accepting these conclusions, but submit that t of dietary restrictions recommended must be assessed by error in each individual case.

As gout, once definitely established, is present in ma latent form throughout the remainder of the patient's necessary to plan the diet having regard to this fact, ar impose too strict a regime. Each case requires individ sideration, depending on whether obesity is or is not pre the ease with which attacks of gout are precipitated.

The following articles should be excluded permanently of their high purine content: Fish-roe, sweetbreads, wh sprats, sardines, herring, mussels, bloaters, heart, kidne goose, and meat extracts. A small to moderate helping should be allowed at one meal and a similar amount of chicken at another. It should be noted that white meat a have as high a purine content as red meat. Fish with a h content, such as salmon, are generally forbidden, but their content is approximately the same as white fish, chicken or while their protein content is lower. Their exclusion can o justified therefore on the ground of a personal idiosyncrasy patient's or because of their fat content. Provided purin protein is taken (*i.e.* milk, cheese or eggs) there is no reas reducing the protein intake, since feeding with purine-free p actually increases uric acid excretion.

In this connection it should be noted that, although leg have a relatively high protein content, they are actually lo purines. Therefore the older conception of the rigid prohibiti legumes in the diet in gout would appear to have no scien basis.

Plenty of green vegetables and fruits, fresh or stewed, shoul taken. Strawberries, spinach and rhubarb are generally prohib because of their tendency to cause oxaluria. If the patient wi to eat them, prohibition should only be rigidly enforced a suitable trials have been conducted. Cereals, milk, cheese, e sugar, jam and honey need not be restricted, for they are fo low or free of purines. If the patient is sensitive to any particu kind of food, it should be prohibited. But the doctor must entir satisfy himself first that a genuine sensitivity exists by caref cross-examining the patient, and, when necessary, by assessing effect of supplying the suspected article of food in a disguised for

tion has already been drawn to the necessity for treating
ty. In cases where the gouty tendency is marked, all forms
cohol should be forbidden. In less severe cases the advice
n on p. 337 should be followed. A diet suitable for chronic
and for the intervals between attacks is given in Diet
31 (p. 493).

Drugs in Gout

e dietetic measures are insufficient by themselves to control
t, it is necessary to discuss the use of drugs.

Treatment of the Acute Attack

far the most effective drug for the relief of the acute attack is
hicum, which when properly administered may be expected
nsure relief within 24 to 72 hours. How the drug produces this
ect is quite unknown. It neither lowers the serum level of uric
l nor increases its excretion. In Great Britain it is the custom
rescribe colchicum in the form of the wine (15 minims) together
h sodium salicylate (20 grains), and potassium bicarbonate (20
ins), the mixture to be taken every two or three hours for the
t twelve hours and thereafter three or four times daily until the
ite symptoms have subsided. Leading American authorities
ench, Bauer and Klemperer, 1944) state that more consistent
l effective results are produced if the drug is prescribed in the
n of crystalline colchicine, since tinctures and wines are notori-
ly unstable preparations and variable in their content of the
ive principle. They recommend that colchicine be prescribed
the form of pills or capsules each containing half a milligram
ain $\frac{1}{120}$) of the alkaloid. The initial dose recommended is
nilligram followed at two-hourly intervals by half a milligram
til relief of pain is obtained or vomiting or diarrhœa result.
ovided the patient is kept in bed, pain should be relieved within
o 72 hours. The total amount of colchicine to relieve the acute
ack varies from case to case, but in general is somewhere
tween 4 and 8 milligrams. In some cases the gastro-intestinal
nptoms may be so severe as to require treatment with Tinctura
pii Camphorata (60 minims after each loose motion). In the
eatment of subsequent acute attacks of gout the total amount
colchicine prescribed should be 1 or 2 milligrams less than that
antity found to produce severe diarrhœa. So successful may
is method be that it is recommended that patients should always
rry with them a supply of colchicine pills which should im-

mediately be taken in the way recommended above, on the appearance of prodromal symptoms or the first twinge of articular pain. By this means it is claimed that patients may be spared prolonged and incapacitating seizures.

Treatment between Attacks

Bauer and Klemperer's (1944) metabolic studies have shown that sodium salicylate and acetylsalicylic acid are as effective as cinchophen in promoting the excretion of uric acid, and hence are the drugs of choice for this purpose since they do not possess the toxic properties of cinchophen. The dose of sodium salicylate is 100 to 120 grains and of acetylsalicylic acid 60 to 70 grains daily. Alkali should be prescribed with each dose of sodium salicylate. It has been the custom in the past to prescribe salicylate for three-day periods in each week, but Bauer and Klemperer have shown that the increased excretion of uric acid resulting is followed by an increased retention so that the extra amount excreted over a period of seven days is insignificant. Accordingly, they recommend that when attacks of gout are occurring rapidly at short intervals, salicylate in the doses suggested above should be given daily for a month, by which time the concentration of serum uric acid will be reduced to one-third of the pre-treatment level, and its return to the original level will be delayed for several weeks.

Opinion is still divided as to whether or not cinchophen should be used in the treatment of gout. It is well known that it increases uric acid excretion and lowers the level of the blood uric acid, but some patients have an idiosyncrasy to the drug, and a serious, even fatal, hepatitis may occur in such persons even after only small amounts of cinchophen have been taken. The dangers of toxic reactions have been probably overstressed if the measures mentioned below are carried out. Any evidence of previous or present liver disease is, therefore, an absolute contra-indication to its use. It should be withheld from elderly patients suffering from cardio-vascular or renal disease. Cinchophen is probably best reserved for chronic cases in which dietetic treatment, plus sodium salicylate, is not effective in lowering the blood uric acid or preventing acute exacerbations. When it is decided to use the drug, it should be prescribed in 7½-grain doses three times daily, along with 30 grains of potassium bicarbonate, for three or four days, and then stopped for at least a week. Ample sugar or glucose, and plenty of fluid, should be taken during the period in which the drug is being administered. Cinchophen should be discontinued when the blood uric acid reaches a normal level.

RHEUMATISM: GENERAL CONSIDERATIONS

While gout is the one form of arthritis in which dietary measures form the mainstay of treatment, modifications of the diet are of importance in the rheumatic diseases – rheumatoid arthritis, osteoarthritis and fibrositis.

General Principles

In our opinion there is no particular type of diet which can be prescribed routinely for all the different conditions which are grouped together under the term 'chronic rheumatic diseases'. Each case must receive individual dietetic treatment according to the patient's condition, *i.e.* whether stout or thin, weak or strong, febrile or afebrile, etc., and according to his capacity to digest the various articles of food allowed. No article of food should be forbidden because of some fad of either the doctor or the patient but only when there is sound evidence, clinical or experimental, to support the restriction ordered.

The view has long been widely held, both by the laity and by many members of the medical profession, that sufferers from the chronic rheumatic diseases have inherited or acquired what is vaguely called an 'acid diathesis'. Accordingly some authorities have restricted fruit from the diet on the erroneous basis that the acids of fruits tend to increase the patient's acidity. Others would restrict protein and purine foods because of their richness in cell nuclei from which purine bodies and ultimately uric acid are derived. As has already been pointed out it is only in true gout that purine metabolism is at fault. Imbued with the theory of the 'acid diathesis' some dietitians lay great stress on avoiding or severely restricting the foods which yield an acid ash. To influence the acid-base balance materially by diet it would be necessary to exclude: 1. bread, flour and cereals; 2. meat, chicken, rabbit, pork, and bacon; 3. fish, eggs, and cheese. The diet allowed would therefore consist of fruit and vegetables, milk, cream and butter, sugar and jam. There are several reasons why attempts at influencing acid-base metabolism by diet are not worthy of serious consideration: (i) we know of no satisfactory evidence that the acid-base metabolism is altered in the chronic rheumatic diseases; (ii) when asthenia and wasting of the muscles is a prominent feature, as in rheumatoid arthritis, satisfactory repair and replacement demand adequate amounts of first-class protein in the form of meat, chicken, fish and eggs – food which are also of particular value for the prevention and correction of anæmia; (iii) a diet constructed essentially from the foods yielding a basic ash is extremely limited in type and therefore monotonous; (iv) if it is desired to influence the acid-base balance, it can be more effectively and easily done by permitting the patient to eat an ordinary diet and prescribing alkalis by mouth, *e.g.* potassium citrate 60 grains three or four times a day.

Mattice (1941), in an excellent review of the acid-base factors in nutrition, gives a clear account of the difficulties and fallacies involved in the alteration of the *p*H of the urine by dietary means.

RHEUMATOID ARTHRITIS

Since the origin of this disease is probably infective, dietary treatment is not likely to have any specific curative action. The importance of the diet in rheumatoid arthritis lies rather in the fact that an adequate diet will tend to correct the under-nutrition so common in this disease and thus raise the general resistance of the patient. The diet should be built up on the following principles :

1. *An Adequate Caloric Intake.* As a general rule, sufferers from rheumatoid arthritis are thin and are under their ideal body weight. Therefore, a diet of high caloric value should be prescribed, bearing in mind that patients in the more acute stage often suffer from anorexia and dyspepsia, and may not be able to take a full diet. In this type of case Pemberton and Osgood (1934) claim good results following drastic reduction in the food intake, nothing but orange juice being allowed for the first two or three days of treatment. The diet is then gradually built up by the addition of protein and fat in the form of milk, butter and eggs. Such a regime should not be employed in febrile cases or in those who are markedly under-nourished. For such cases, small feeds of high caloric value can be given in the form of a light cereal (Benger's, Allenbury's, Cream of Wheat, Farex, etc.), made up with milk and a little cream and sweetened with lactose; switched egg in milk, milk cocoa, etc. (see Diet in Fever, p. 498).

Certain cases of rheumatoid arthritis are met with, however, in which there is little or no asthenia and in which the body weight may even be excessive. The patients are fat and flabby. A reducing diet on the lines described on p. 350 should be prescribed and attention be given to maintaining an adequate supply of grade A protein of high biological value, vitamins and minerals.

2. *Moderate Restriction of the Carbohydrate Intake.* On the ground that carbohydrate metabolism is defective in rheumatoid arthritis owing to defective capillary circulation and muscular atrophy, certain authorities recommend that some restriction of the carbohydrate intake should be made by limiting bread and other starchy foods, and forbidding sweetmeats. If this is done, the caloric value of the diet must be maintained by a liberal intake of fatty foods – cream, butter, vitaminized margarine and cheese.

3. *A liberal Protein Intake.* Another fallacy that must be exposed is the belief that animal protein is harmful in rheumatoid arthritis. This idea probably dates from the time before gout was separated ætiologically from the chronic rheumatic diseases. In a wasting and often febrile disease like rheumatoid arthritis, a liberal intake of first-class protein is essential to replace the body tissues broken down.

4. *A liberal Vitamin Intake.* Although there is no satisfactory evidence that vitamin deficiency is the cause of rheumatoid arthritis, nevertheless, once the disease is established, a relative avitaminosis may develop as in many toxic or infective states. Therefore, in an endeavour to improve the general condition of the patient and increase his resistance an adequate intake of vitamins must be secured. To ensure an ample supply of vitamin C, plenty of fresh fruit and vegetables should be taken. The old prejudice against the eating of fruits in rheumatism owing to their acidity has nothing to support it, for the fruit acids are absorbed as salts – citrates, malates and tartrates – and the acid radicle is oxidized in the body leaving the basic radicle, so that fruits actually have base-forming properties. Vitamin B_1 requirements are also increased under conditions of enhanced metabolism, and additional supplies should be given, particularly if the appetite is impaired and an atonic condition of the muscle of the intestines is present as is the case in rheumatoid arthritis. Accordingly the diet should provide wholemeal cereals (if the patient's digestion permits), eggs, liver, etc. (see p. 46), to which should be added a supplement of some preparation rich in the vitamin, such as yeast or yeast tablets, Marmite or Bemax.

The fat soluble vitamins are of equal importance, vitamin A for its protective action on epithelial membranes and the increased resistance to infection which this gives, and vitamin D for its action in promoting calcium absorption and so combating the skeletal decalcification so common in rheumatoid arthritis. Vitamins A and D can be supplied as cod- or halibut-liver oil or any of the proprietary concentrates mentioned on page 99.

5. *A liberal Intake of Calcium and Iron.* Skeletal decalcification is, as we have just stated, often a prominent feature of the clinical picture of rheumatoid arthritis. The cause of this demineralization is not fully understood, but it is logical to attempt to treat it by giving the patient a diet with a liberal calcium intake, which implies at least 1½ pints of milk daily, plus cheese if possible. Vitamin D administration may increase the absorption and retention of calcium. Anæmia is another common feature of rheumatoid arthritis. While the diet should contain plenty of iron-rich foods such as meat and green vegetables, the administration of medicinal iron will also be required in the presence of an established anæmia. A series of small blood transfusions may be of real value for markedly debilitated patients.

To sum up, a well-balanced, bland, nutritious diet should be prescribed, of sufficient caloric value to maintain the patient's weight at or near normal level, and containing optimal amounts of minerals and accessory food substances. The articles of diet should be varied as much as possible and served in an attractive manner. The three main meals should be small, and intermediate feeds of milk or one of the proprietary invalid foods (Benger's, Farex Ovaltine, etc.) should be given.

OSTEOARTHRITIS

This is a degenerative condition, probably of constitutional origin, but precipitated by factors such as injury to a joint, occupational strains, etc. Osteoarthritis is especially prone to occur in the so-called sthenic type of patient, with a stocky build and a tendency to obesity. Obesity is an important factor causing chronic joint strain through faulty posture and undue pressure on articular surfaces. Therefore, in both the prophylaxis and the treatment of osteoarthritis, the treatment of obesity is of the greatest importance. This is dealt with in detail on p. 349. In the special form of osteoarthritis known as chronic menopausal arthritis, which affects the knees and begins as a proliferative synovitis, thyroid extract should be given as well.

FIBROSITIS

The diet prescribed in fibrositis will depend on the ætiology of the condition in each individual case. Where focal sepsis, occupation, injury or chronic muscular strain is the primary cause, diet will play a minor part. In a proportion of cases a careful history will reveal that the onset of the acute attack follows some dietetic indiscretion. The underlying defect in such cases is not clearly understood, but it may be the inability of the liver to complete the metabolism of purine bodies, or a failure of digestion leading to the absorption of toxic products from the gut. In every case of fibrositis a careful inquiry should be made into the dietary history, and suspected articles of food or drink should be forbidden. It should be remembered that occasionally, in the more chronic type of case, true gout may be the underlying cause.

During the acute attack the diet in all cases should be light and lacto-vegetarian in nature. Large quantities of fluids should be drunk, at least 6 to 8 glasses daily. In chronic cases, even where the evidence of a gouty basis is lacking, it is generally advisable to prohibit the purine-rich foods (liver, kidneys, sweetbreads) and restrict animal proteins to a moderate degree. Starchy foods, such as pastry, suet puddings and bread, should also be restricted, especially if obesity is present. Beer and sweet wines are better avoided. Plenty of fluids should be drunk, and constipation corrected, if necessary, by liquid paraffin. When there is a true gouty diathesis, treatment should be advised along the lines indicated on p. 337.

CHAPTER XVI

OBESITY AND EXCESSIVE LEANNESS

OBESITY: GENERAL CONSIDERATIONS

WHILE it is easy to define obesity as an excessive accumulation of fat in the storage areas of the body, it is difficult to state in individual instances the point at which this accumulation may be considered to be excessive. It must not be forgotten, too, that certain well-marked changes in weight occur so commonly as age advances that they can be accepted as physiological. Most individuals gain at least seven pounds in weight during the years from 30 to 40, while an even greater gain may take place in the succeeding decade. After the age of 50 to 60 there is usually a decline in weight. Obesity, as a condition requiring treatment, can be taken to be present when fat deposition has led to diminished well-being of the individual or to diminished functional activity.

Harmful Effects of Obesity

It is not on æsthetic considerations that a serious view is taken of the development of obesity but on a realization of the multiple harmful effects which long-continued obesity exerts on the various systems. Among these may be mentioned:

1. *Decreased Longevity over the Age of Forty.* Life Insurance statistics show that an excess of body weight of even a moderate degree decreases the expectancy of life after the age of 40.

2. *Interference with Physical Activity.* The extra body weight of itself tends to reduce the individual's activity, and to this there are usually added orthopædic disabilities due to excessive body weight and the resultant postural defects. Flat-foot and arthritis are common complications of obesity.

3. *Effects on the Circulation.* The work of the heart is increased by the extra mechanical work entailed in moving the overweight body, while the efficiency of the heart in responding to this call is lessened by the deposition of fat in and around the myocardium. It is, therefore, not surprising that myocardial degeneration is frequent among the obese.

4. *Predisposition to Disease.* Obesity appears to be a factor of ætiological importance in diabetes, gout, cholecystitis, hypertension, and possibly other diseases.

5. *Interference with Skin Functions.* The excessive deposit of

subcutaneous fat interferes with the regulation of heat loss from the body. Also, there is an increased tendency to skin infections in the obese, especially at the flexures.

A long list of diseases could, therefore, be given, including such conditions as diabetes, gout, gallstones, hypertension, heart disease, arthritis and postural backache, in which the correction of any obesity present constitutes an important part of the treatment of the presenting disease.

Classification of Obesity

Any attempt to subdivide obesity is unsatisfactory, since the pathogenesis of the condition is not properly understood. At one end of the scale are the cases due to some obvious endocrine disturbance, while at the other end are those in which caloric intake has clearly been excessive in relation to physical activity. These two groups probably include only a small minority of the cases of obesity, however, and therefore any classification must include a third subdivision of unknown ætiology and usually referred to as constitutional obesity.

Exogenous Obesity

Certain authorities consider that there is no metabolic abnormality in obesity, and hold that it is caused by a food intake in excess of the metabolic requirement, due either to perverted habit or chronic alcoholism, or to a failure to reduce the diet as activity decreases with age. While some cases can certainly be attributed to these exogenous factors, there still remains the question why some persons can eat large amounts of food without developing obesity.

Endocrine Obesity

This group includes obese individuals who show other signs of endocrine disturbance, such as:

1. Pituitary diseases – pituitary basophilism (Cushing's syndrome), or genito-pituitary obesity (Fröhlich's syndrome).
2. Thyroid disease. In myxœdema there is usually an increase of subcutaneous fat, and minor degrees of thyroid hypofunction may be sometimes detected in obese patients.
3. Adrenal diseases. Tumours of the adrenal cortex give rise to a picture practically identical with Cushing's syndrome.
4. Gonadal diseases. Obesity is prone to follow deficient function of the sex glands.

Constitutional Obesity

Most authorities relegate the vast majority of obese patients to this group. Such patients eat no more than persons of normal weight, are of normally active habits, and show no obvious evidence of endocrine disturbance. The one positive ætiological factor is that in a great many cases there is a history of obesity in preceding generations. Therefore, it is postulated that such obese patients store fat excessively owing to some as yet unexplained constitutional anomaly of metabolism, which is probably hereditary.

Various explanations for this metabolic anomaly have been put forward. Since a normal person maintains an approximately constant body weight without giving thought to food intake or exercise, it is obvious that there must be some regulatory mechanism. That this mechanism is not controlled by appetite is clear when it is realized that most people eat more than their energy expenditure requires. Nevertheless, in a normal person the excess food is not stored, so that the body must be able, up to a certain point, to burn up with ease any food taken in excess of the energy requirements – the so-called 'luxus consumption' of the foodstuffs. It is postulated that in obese persons this power of 'luxus consumption' is defective, so that food taken in excess of the energy requirements is stored, not burned. This is begging the question, however, as no satisfactory explanation of the mechanism involved can be given. The metabolism of obese persons is not run on more economical lines than that of normal individuals, for the basal metabolic rate in obesity is practically always normal. In fact, this finding suggests that in a patient with constitutional obesity the metabolism of the actively functioning tissues is greater than in a person of normal weight. An obese subject has a larger surface area than normal, and hence if his basal metabolic rate is no lower, his total basal energy production must be greater than that of a normal person. The mass of actively functioning tissues is probably no greater in an obese than in a normal subject, and fatty tissue has a very low metabolism. It would seem therefore that the increased total basal energy production in an obese subject must be due to increased metabolic activity of his actively functioning tissues.

Several workers have claimed to show that the specific dynamic action of the foodstuffs is reduced in obesity. Such a theory would explain the storing of excess food instead of its oxidation, but, unfortunately, most recent work has not confirmed it.

THE TREATMENT OF OBESITY

While the classification of obesity is therefore still unsatisfactory, it is fortunate that as far as treatment is concerned the distinction between endogenous and exogenous obesity is academic, since both forms respond to the same treatment. Whatever the cause, it is clear that in the obese person the intake of food is exceeding, or has at some time exceeded, the energy expenditure, and therapy must be directed towards correcting the balance by decreasing the food intake and increasing the energy expenditure.

A. Reduction of the Food Intake

While a 50 per cent reduction in caloric intake is necessary in most cases, care must be taken that a bizarre or one-sided diet containing an abnormally high proportion of any one constituent is not prescribed. The diet in obesity must fulfil the following requirements :

(i) The body must be kept in nitrogenous equilibrium.
(ii) The diet must contain sufficient bulk so that the appetite is satisfied as far as possible and actual suffering does not occur.
(iii) The diet must meet the mineral and vitamin requirements.
(iv) The loss of weight produced by the subcaloric diet must not be so rapid that the patient becomes weak and exhausted.

Before laying down rigid dietary rules in the treatment of a case of obesity, it is essential to conduct a detailed inquiry into the patient's dietetic habits. 'Hidden Calories' in the form of sweets, excessive ingestion of some specially fattening article of food, or indulgence in between-meal snacks may be revealed in patients who claim to eat no more than a normal person, and in mild cases simple correction of these dietetic errors may be all that is required. Also, it must be remembered that great care is necessary in reducing obesity in the presence of chronic disease such as cardiac disease or hypertension. Treatment should then be so arranged that the loss of weight is more gradual than in the otherwise healthy obese patient. When the excess of weight is only mild or moderate, *i.e.* not more than 1½ stones, reduction of the visible fat (butter, cream, fat meats, pastries, salad oil) and of the concentrated carbohydrates (sugar, bread, cakes and pastries) of the diet may be sufficient.

When the obesity is marked, *i.e.* the patient is two or more stones above the calculated ideal body weight, or if the simple measures described above have been unsuccessful, strict treatment on the lines indicated below must be instituted. Consideration must be given not only to the caloric value of the diet, but also to the relative proportions of protein, carbohydrate and fat in the diet, and its mineral and vitamin content.

The Caloric Intake. It is known that if a normal person is given a diet somewhat under his caloric requirements, there is, for a time, a fall in body weight, which then becomes stabilized at a new level, due probably to a reduction in the basal metabolic rate. The same effect is seen in obese patients on a reducing diet, so that to be effective in producing satisfactory weight loss, the curtailment of the caloric intake must be considerable.

The caloric values of diets used in obesity range from 600 to 1,500. Apart from occasional cases in men engaged in hard physical work, diets of 1,500 Calories usually produce little weight loss, while at the other extreme, diets yielding as little as 600 Calories are too small in bulk and produce weakness and exhaustion. For ambulant patients, diets of 1,000 to 1,200 Calories are most suitable; even a reduction to 1,200 Calories may not produce satisfactory loss of weight except in a very active patient. After the caloric value has been decided on, consideration must be given to the proportion of protein, carbohydrate and fat.

Protein. The intake must be adequate to maintain nitrogen equilibrium, the aim being to obtain a loss of weight by the burning of excess fat and not by the breakdown of body protein. A protein allowance of 0·5 gram per pound of *ideal* body weight is sufficient for this purpose. It can be taken that a total protein intake of about 70 grams will be ample. While nitrogen equilibrium can be maintained on a protein intake considerably less than 70 grams, it is desirable that the reducing diet should contain a relatively large amount of protein, since protein, by its specific dynamic action, stimulates metabolism and hence promotes oxidation of body fat. At least 50 grams of the protein prescribed should be of high biological value, *i.e.* of animal origin (see p. 27).

Carbohydrate. 50 per cent or more of the caloric value of a normal diet is derived from carbohydrate, and dietetic excess in obese patients most frequently takes the form of over-indulgence in concentrated carbohydrate foods. Severe restriction of the carbohydrate quota is therefore necessary in a reducing diet. Limits are set to this reduction, however, by the fact that very marked curtailment of the carbohydrate intake causes weakness, nausea and faintness. Also, the patient burns his own body fat to make good the deficient caloric intake, and if the carbohydrate intake is too drastically reduced, ketosis may develop. Carbohydrate, too, is a more efficient protein sparer than fat.

In a 1,000 Calorie diet, the carbohydrate quota should comprise 100 to 120 grams. It is important that a large part of this quota should be supplied by fresh fruit and vegetables. These have a low caloric value, so that generous amounts can be prescribed without materially increasing the caloric intake, and their bulk will satisfy the appetite and tend to correct the constipation from which so many obese patients suffer and which is apt to be increased by a low caloric diet. Fruit and vegetables also form good sources of vitamins. The intake of bread, cereals and potatoes must be strictly limited. The concentrated carbohydrate foods such as sugar, preserves, tinned fruit, and pastries must be prohibited, as well as sweetened drinks and alcohol. The last is important, as a considerable increase in caloric intake may occur unwittingly from this source; thus, a single glass of sherry adds about 50 Calories to the diet, one small whisky almost 100 Calories, and a glass of beer no less than 120 to 150 Calories (see Table 23, p. 445).

Where the prohibition of sugar imposes hardship, saccharin can be used as a sweetening agent as described on p. 236.

Fat. As the patient will use his own body fat, the amount of fat in the diet can be reduced to a minimum, although a certain small amount is desirable to make the diet palatable. In a 1,000 Calorie diet, where 70 grams of protein and 100 grams of carbohydrate are being given, the fat quota can only be about 30 grams. In such a diet, the use of skimmed milk in place of whole milk will ensure a reasonable supply of minerals without adding to the fat intake. A small portion of butter or vitaminized margarine is best included to make the patient's bread ration more palatable. The drastic reduction in the fat allowance reduces the intake of fat-soluble vitamins, and it is probably advisable to prescribe one of the vitamins A and D concentrates such as Radiostoleum or Adexolin.

A suitable obesity diet of 1,000 Calories, embodying the principles described above is given in Diet No. 32, p. 495.

Should it be desired to increase the diet to 1,100 or 1,200 Calories, this can be done by the addition of one or two 100 Calorie portions of protein or carbohydrate as described on p. 443.

Table 22 (p. 444), which gives the principal foodstuffs in descending order of their caloric value, will help the practitioner to decide what foods should or should not be allowed. Obviously, the majority of the articles in a 1,000 Calorie obesity diet must be drawn from the lower half of the table.

Water and Salt Restriction in Obesity. The question is oft raised as to whether fluid restriction should be practised during th treatment of obesity, since there is evidence that some obe patients have a definite tendency to retain water. It is probabl unnecessary to curtail fluid intake except in cases where an un satisfactory weight loss occurs on a low caloric diet which th patient is definitely not exceeding, or where there are speci indications, *e.g.* oedema.

Reduction of the salt intake is of value, and the patient shou be instructed to use as little salt at table as possible.

Results of Dietary Treatment. As in any dietetic treatment wher the patient is entrusted with the carrying out of the regime pre scribed, the success or failure of a reducing diet in obesity wi depend to a large extent on the intelligence and co-operation of th patient. To impress on the patient the paramount importance o strict dieting in the treatment of obesity, he should be made t weigh or measure all the articles of the diet until he has becom proficient in judging their weight by eye. At this point special car is needed, and if satisfactory weight loss ceases, it can be suspecte that the patient is being unduly generous in his helpings.

The rate of weight loss aimed at is about 2 lb. per week. Ther will be a greater loss in the first week or two, probably owing t fluid loss. If the subsequent weight loss exceeds 2 to 3 lb. a week the diet must be increased slightly. Weight reduction is a matte of months not weeks, and there is no advantage in rapid weigh loss, which will probably weaken the patient unduly. If the weekl loss is less than 2 lb., and this is more frequently the case, th patient must be thoroughly questioned as to the closeness wit which he is following the prescribed diet, since poor co-operation i the commonest cause of an unsatisfactory rate of weight reduction Especially important is a searching inquiry into the partaking o any extras such as sweets or between-meal snacks, in which th patient may indulge through ignorance or lack of co-operation.

The patient must be seen frequently by his doctor, especiall during the early stages of treatment, as he will need encourage ment, advice, and possibly reprimand. The weight should not b taken more than once a week. Occasional fluctuations of weight probably due to changes in the fluid content of the body, some times obscure the true rate of loss of weight, and it should b remembered that there is frequently, in normal as well as in obes women, a gain in weight just before menstruation, followed by a loss of weight during the period.

As the progress of an obese patient on a low Calorie diet is followed, it will usually be found that the rate of loss of weight becomes steadily less, until the weight becomes stabilized, sometimes still well above the ideal. This is probably due in part to a fall in basal metabolism mentioned already as a consequence of the reduction of the food intake, and it is justifiable to give the patient thyroid at this point, if further reduction of weight is desired.

After Treatment. Where the patient's weight has been reduced either to the ideal or to the greatest extent possible with dietary measures and possibly thyroid as well, the question arises as to what subsequent steps, if any, are necessary in order to prevent his again becoming obese. If treatment is entirely discontinued, most patients will begin to lay down excess fat again; hence a modified dietary regime will probably be necessary for life. This does not entail a permanently strict diet. It is usually sufficient to reduce the intake of the visible fats (butter, cream, fat meats, salad oil, etc.) and concentrated carbohydrate foods (sugar, cake, pastries, bread, preserves, etc.). A minority of cases, usually those with other evidence of endocrine disturbance, will tend to become rapidly obese again as soon as the strict diet is stopped, and in these a permanent adherence to the rigid diet is, unfortunately, necessary if the good results of reduction treatment are not to be undone.

Intensive Cures. Some workers have advocated the use of diets containing as little as 600 to 800 Calories, but such intensive treatment is only called for when ambulant treatment on a 1,000 Calorie diet has not secured satisfactory weight loss or when there is gross obesity. We do not advocate the use of diets containing less than 800 Calories, and even on this amount patients often complain of weakness and faintness. Such a diet must not be continued for more than three weeks, after which it must be raised to 1,000 or 1,200 Calories. The patient is best kept in bed, certainly for the first week and if necessary during the whole course. Loss of body protein must be prevented by prescribing 0·5 gram of protein per pound of body weight; the remainder of the diet is made up of carbohydrate derived from fruit, vegetables and a small ration of bread, with an unavoidable minimum of fat (about 20 grams). Fat-soluble vitamins must be supplied in the form of one of the A and D concentrates. An 800 Calorie diet designed on the above lines will be obtained by omitting the butter from the 1,000 Calorie obesity diet, No. 32 (p. 495).

B. Measures to increase Energy Expenditure

If the energy expenditure of an obese patient on a low Calorie diet can be increased, there will be increased combustion of foodstuffs and, therefore, an increased utilization of body fat.

This increased energy expenditure can theoretically be obtained in two ways – by physical exercise or by the use of drugs stimulating the metabolic processes. Before going any further, we must emphasize that, as practical measures in the therapy of obesity, both these aids to energy expenditure are of much less importance than the dietetic treatment described above.

1. *Physical Measures*

It is a popular idea that obesity can be effectively combated by increasing the amount of muscular exercise taken by the individual. However, quite apart from the fact that the obesity of itself will probably debar the patient from engaging in any but the milder forms of exercise, it is only by severe and long-continued muscular exercise that a significant increase in energy expenditure can be attained. The maximal energy expenditure possible over short periods of a few minutes by trained athletes is in the region of 600 Calories per hour – a burst of such energy expenditure over fifteen minutes would consume 150 Calories, which in terms of fat is just over ½ oz. Milder exercise has even less effect, and a person of normal weight walking along a level road only expends about 60 extra Calories per mile. Another factor which makes exercise of questionable value in the treatment of obesity is its effect of increasing the appetite and so tempting the patient to break his rigid diet and probably add many more Calories to the balance than were subtracted by the exercise. If the obese patient who played a vigorous game of squash racquets for a quarter of an hour quenched his thirst with one glass of beer, he would have replaced all the Calories lost through his well-meant exertions. Similarly the good effects of walking a mile would be more than counterbalanced if the patient ate an extra ounce of bread on his return.

These facts and figures are not quoted with the object of denying that physical exercises may aid in weight reduction or of decrying its value in the promotion of health. They are given with the object of impressing on the practitioner the inadvisability of relying solely or mainly on exercise as a means of weight reduction.

Hot baths and vapour baths are of no use, any reduction of weight being due to loss of body water which is rapidly replaced by fluids drunk by the patient. Similarly, massage does not increase the energy expenditure of the patient, only that of the masseur. Cold baths undoubtedly stimulate metabolism, and may be employed in the exceptional cases who can tolerate them. They can scarcely be considered as a practical measure and are absolutely contra-indicated in the presence of any signs suggesting cardiac disease.

2. *Drugs stimulating Metabolism*

1. *Thyroid.* The effect of this hormone is to increase the general metabolism, and this action led to its introduction in the treatment of obesity.

The question whether or not thyroid should be used in obesity not associated with hypothyroidism is still the subject of controversy. Some workers, arguing on the basis that the basal metabolic rate is not lowered in obesity, consider that loss of weight following the use of thyroid is an indication of toxicity. Others have claimed good results in selected cases.

Most obese patients can have their weight reduced satisfactorily without the use of thyroid. Its promiscuous use is, therefore, to say the least, unnecessary, but it is of undoubted benefit in certain cases:

(i) In frank myxœdema, and in obese patients with signs suggesting hypothyroidism such as dry skin, slow pulse, loss of hair, and subjective feelings of cold.
(ii) When the rate of weight loss in an obese patient being treated by dietary measures has slowed down and stopped at a body weight still too far above the ideal.
(iii) In obese patients on a low Calorie diet who complain of undue sensitivity to cold such as a low Calorie diet sometimes induces.

Except in frank myxœdema, the initial dose of thyroid should be small, say ¼ to ½ grain of thyroideum (B.P.) twice daily, increasing later to 1 grain twice or thrice daily if necessary. The dosage is regulated chiefly by the pulse rate, and the resting pulse rate should not be allowed to exceed 90. If tachycardia of greater degree or other signs of toxicity such as tremor, nervousness, palpitation, etc., develop, the drug must be withdrawn or the dose reduced. Patients being treated with thyroid must be under frequent observation by their doctor so that the earliest symptoms of toxicity can be recognized.

2. *Dinitrophenol.* This drug stimulates metabolism by increasing carbohydrate oxidation, and was introduced some years ago as a

cure for obesity. It should never be employed owing to its marked toxic action, the commonest features of which are hyperpyrexia and hepatitis, which may prove fatal.

LEANNESS AND UNDER-NUTRITION

A person more than 14 lb. under his ideal weight may be considered lean. The condition of leanness must be distinguished from emaciation (loss of weight through disease) and inanition (loss of weight due to deficient food intake). It is a personal characteristic in many cases, compatible with perfect health. Two types of leanness can be distinguished:

1. *Sthenic Type.* Persons of this type have normal stamina and emotional poise. The weight is maintained at a constant level over long periods, and the condition can be regarded as a metabolic tendency opposite to that postulated in constitutional obesity – the ‘ luxus consumption’ of foodstuffs is set at a high level, so that all surplus nutritive material is burned up very efficiently. This type of leanness requires no treatment.

2. *Asthenic Type.* Individuals belonging to this group lack stamina and physical vigour, have narrow chests and usually visceroptosis, and show emotional instability. There is often a complaint of vague dyspepsia, and a vicious circle is set up, the patients becoming afraid to eat so that actual under-nutrition is added to the constitutional leanness. An extreme example of this is:

Anorexia Nervosa. This condition, most common in adolescent girls and young women, is a functional condition in which there is complete loss of appetite of psychogenic origin, usually relating to some factor such as unhappiness at home or work, an unhappy love affair, and so on. The lack of appetite and consequent abstention from food is an hysterical symptom, and the dislike for food may amount to actual loathing. If unchecked, extreme physical weakness, emaciation, and even death may follow.

If the weight can be increased in the asthenic type of leanness, there is usually an improvement in the general health.

Treatment

This is essentially the opposite of the treatment of obesity as far as diet is concerned, but the patient's psychological attitude to food must be corrected.

1. *The Patient's Distaste for or Fear of Food must be overcome*

The patient should be removed from home surroundings and treated in a hospital or nursing home, as she will certainly not take all the food prescribed if left to her own devices. The patient's symptoms should not be disregarded, and her doctor should explain that it is only by eating that the desire for food will return. At the same time great firmness is required, and no attention should be paid to the patient's whims regarding certain foods, except in the rare cases with definite contra-indications due to food allergy. Meals should be served at regular intervals, and the patient told she must eat all the food set before her. It may be necessary at first for the doctor to remain with the patient during meals, when resistance can be broken down by firm but tactful handling. In anorexia nervosa, however, no dietetic treatment is likely to be successful unless the psychological factor can be discovered and remedied.

2. *The Total Food Intake must exceed the Maintenance Quota*

The total caloric value of the diet should be up to 50 per cent above the maintenance requirement as calculated from the patient's ideal weight. The constituents of the diet require some consideration:

Protein. The quota should be 100 to 120 grams, at least 50 per cent of which should be of animal origin.

Carbohydrate. The diet should contain only small quantities of fruit and green vegetables, as their bulk may prevent patients taking food of higher caloric value. Carbohydrate should be given in concentrated forms – cereals, potatoes, preserves, sugar. Fruit juices should be included, so that an adequate intake of vitamin C is secured.

Fat. Owing to its high caloric value, the fat quota should be liberal, but care must be taken not to force fats to the point of nausea. Cream, butter, eggs, bacon and olive oil are the forms in which fat should be prescribed. Butter or vitaminized margarine is especially useful as it can be spread thickly on bread, or added to potatoes, or utilized in cooking, and so administered in large amounts without tending to produce nausea.

Milk and cream are the foods on which greatest dependence should be placed, as they can be forced owing to their fluid character, lack of bulk and absence of strong taste. Milk alone is not a concentrated food as 1 oz. yields only 17 Calories. However, a mixture of equal parts of milk and 20 per cent cream (40 Calories per oz.) makes a drink which is not too fatty, and many Calories can be administered in this way; or the caloric value of milk can be increased by the addition of lactose which can be added in comparatively large amounts (2 to 3 oz. to the pint of milk), owing to its relative lack of sweetness. Eggs are also a valuable source of Calories and may be combined with milk as egg-flip, custard, etc.

In the arrangement of the meals, it is probably best to employ the method of giving small but frequent feeds rather than setting the patient down to a very large meal thrice daily. When the lack of appetite is severe, as in anorexia nervosa, a start may be made with two-hourly fluid feeds as in the Diet No. 33 (p. 497). As soon as the patient can cope with this diet, it must be increased by the addition of solid foodstuffs, and the meals arranged as three main meals plus intermediate feeds.

3. *Insulin may be given*

One of the prominent symptoms of hypoglycæmia is a sensation of hunger. Therefore, many workers recommend the use of insulin to induce hunger sensations in patients with the asthenic type of leanness, so that their appetite is improved and the food intake correspondingly increased. The usual dose is 10 to 20 units given half an hour before breakfast and the evening meal. Sweets, sugar or some other concentrated carbohydrate food should be given during the forenoon and in the late evening to prevent the onset of hypoglycæmia in the middle of the forenoon or at night. While this treatment is often effective, it is uncertain whether this is actually due to the insulin or merely to the psychological effect of the injection.

TREATMENT OF MALNUTRITION DUE TO STARVATION

The value of casein hydrolysates in restoring and maintaining nitrogenous equilibrium in patients suffering from the various medical and surgical conditions described on p. 389, naturally directed attention to their possible uses in the treatment of malnutrition due to starvation. A preliminary report from India on the treatment of a small series of patients dying from starvation in Bengal in 1943, with a papain enzyme digest of meat, claimed encouraging results. It was believed that the basic ration in certain parts of Holland under German occupation was as low as 400 Calories daily and it was assumed that when liberation occurred large numbers of persons suffering from extreme emaciation would be encountered. Accordingly, the authorities concerned requested the Medical Research Council to arrange for the production of casein hydrolysates on a large scale. It was decided that for intravenous therapy acid hydrolysates should be manufactured, tryptophane being added subsequently. This decision was based mainly on the difficulty of manufacturing large quantities of enzyme digests which were free from pyrogens produced by bacterial growth during the period of incubation. Enzyme digests of casein were, however, made on a large scale for oral use. Units containing hydrolysates for both intravenous and oral use, glucose, saline and the apparatus required for administration, with directions for use, were assembled and packed. Before the liberation of German-occupied Holland, certain concentration camps in Germany were overrun by the advancing Allied armies, in which were found indescribable conditions of disease and malnutrition. The worst of these was the horror concentration camp at Belsen which the senior author had the opportunity of visiting for several days. Tens of thousands of internees died of starvation and disease in the period immediately preceding and subsequent to liberation, and some sixteen thousand survivors were hospitalized in a German barracks which was adjacent to the concentration camp. The patients were suffering from every degree of malnutrition. Frank vitamin deficiency diseases such as scurvy, beri-beri and pellagra were conspicuously absent. The daily ration of vegetable soup apparently supplied the vitamin C requirements of the internees, while the vitamin B content of the coarse black bread was sufficient to meet the vitamin demands of individuals whose diets were less than 800 Calories daily in the majority of cases. The principal clinical features encountered were gross emaciation, anæmia, cardiac weakness, famine œdema due to hypoproteinæmia, dehydration, hæmoconcentration and low blood volume due to persistent diarrhœa and to fluid deficiency, no water being available for drinking purposes for several days prior to liberation. The clinical picture of malnutrition was further complicated by co-existing infectious diseases such as typhus fever, enteric fever, dysentery and tuberculosis. Unfortunately, this unique opportunity of deciding, under controlled conditions, the best way of treating patients dying from famine could not be fully utilized because of the multitude of patients and the great scarcity of doctors, nurses, equipment and laboratory facilities. In addition, language difficulties and lack of co-operation of patients, due to fear that any special methods of feeding or the giving of intravenous injections were a prelude to their transference to the torture chamber and the crematorium, were further serious handicaps.

The possibilities for accurate study of the incidence, clinical features and treatment of starvation in Western Holland, following liberation from the Germans, were greater than at Belsen for the following reasons: (1) Nutritional survey teams, specialists in nutritional problems, and special medical feeding units were assembled in readiness to start work immediately liberation occurred. (2) Stocks of special foods were imported and were available in ample amounts for immediate use. (3) Properly staffed and equipped hospitals were available for studying the problems of starvation. (4) The complicating feature of infectious diseases, which was such a prominent feature at Belsen, was largely absent. The incidence of under-nutrition was assessed by the survey teams, and it was calculated that approximately 200,000 persons in Western Holland were suffering from starvation (5 per cent of the total population of the area). The cases were largely confined to the large cities, and the most serious ones were found in the older-age groups. Only the worst cases approximated to the picture present at Belsen, and these fortunately were seldom suffering from co-existing serious infectious diseases. The clinical features present in Western Holland were similar to those described at Belsen. It was noteworthy that in Holland, as in Belsen, classical examples of vitamin deficiency diseases were extremely rare.

Reports on the treatment of starvation in Belsen and Holland have now been published by Lipscome (1945) and by Burger, Sandstead and Drummond (1945), respectively. The four most important measures in the treatment of large numbers of starving persons are the provision of rest in bed to conserve body heat, good nursing, the treatment of infection and the prevention of its spread and suitable feeding. It is with this last measure that we are particularly concerned.

It has long been known from experiments on animals that the processes of digestion and absorption from the alimentary tract are seriously affected by continued starvation. Structural changes of the columnar epithelial cells of the intestinal mucosa result, which expose the intestines to irritation by undigested food with consequent diarrhœa. There also existed a clinical belief that in the final stages of starvation patients were so weak that their capacity to swallow was largely or completely lost. Accordingly, it was assumed that parenteral feeding, or alternatively gavage, appeared to offer the only means of supplying nutriments to such patients. In practice, however, it was found that patients were able to sip and swallow fluid to within a few minutes of life becoming extinct, and that even those who were desperately ill were able to digest and absorb skimmed milk and glucose saline far better than was anticipated. The fact that at Belsen severe diarrhœa due to infection or malnutrition was an almost constant feature of the late stages of starvation, suggested that in such patients preliminary treatment by parenteral feeding methods would be required.

Parenteral Feeding Methods

1. *Intravenous Casein Hydrolysates*

Lipscome states that the intravenous injection of casein hydrolysates was only used in a small number of cases at Belsen because the 5 per cent solution of the acid hydrolysates available caused venous thrombosis, while the volume of a 2½ per cent solution required to produce sufficient nitrogen increased œdema and ascites and risked overloading the circulatory system. In addition, the dearth of trained personnel experienced in parenteral therapy militated against the proper trial of intravenous methods. Burger, Sandstead and Drummond state that the use of intravenous casein hydrolysates had to be abandoned in Holland because of the venous thrombosis produced. The value of amigen, the American preparation of casein hydrolysates prepared by enzynic digestion, in a wide variety of conditions, has been described in detail on p. 389. Accordingly, it would be unwise to dismiss this form of therapy as valueless in the treatment of severe starvation, particularly if diarrhœa is an important feature, because of the unfortunate effects produced by the irritating acid hydrolysates employed in Holland and Belsen. It should be noted that Janet Vaughan has reported that of five patients who were suffering from severe starvation at Belsen, four responded well to the intravenous infusion of amigen. Further carefully controlled investigations are obviously necessary before a final decision can be reached on the place of intravenous hydrolysates in the treatment of starvation.

2. *Blood and Plasma*

Because of the multiplicity of factors which may be important in the causation of the shock or collapse seen in the final stages of starvation, great difficulties exist in deciding on the material which should be given intravenously and the quantities to be administered. It was strongly emphasized by workers at Belsen that cardiac weakness was so marked in many cases that the introduction of even a litre of fluid intravenously might lead to left ventricular failure, pulmonary œdema and death. Many patients were so dehydrated that famine œdema was not present initially, but when an attempt was made to deal with the dehydration, subcutaneous œdema, ascites and pleural effusion developed. Moreover, the marked hypoproteinæmia present (the mean plasma albumin in ten serious cases at Belsen was found by Janet Vaughan to be 2·09 grams per 100 c.c.) was further aggravated when intravenous infusions of casein hydrolysates or glucose saline were given and in consequence an increase in tissue œdema resulted. In these circumstances it was obvious that there was a considerable danger in administering intravenously more than one litre of 5 per cent casein hydrolysate in 10 per cent glucose saline in 24 hours, a quantity which was certainly insufficient to restore nitrogenous equilibrium or materially affect the energy requirements. If emphasis is placed initially on restoring the blood volume, and at the same time giving the maximum of protein with the minimum of fluid intravenously, then serum or plasma has obvious advantages over casein hydrolysates. Transfusions of 1,000 c.c. of single strength serum, or 500 c.c. of double strength, were successfully employed, additional fluid by mouth being allowed. Further experience may show that in the advanced stages of starvation with co-existing diarrhœa, the initial treatment may be by plasma or serum transfusions for the first 24 to 48 hours, to be succeeded thereafter by intravenous casein hydrolysates. Blood transfusions were of real value in combating severe anæmia, which was found to be extremely resistant to iron, liver extract and vitamin C, but slowly responded to a high protein diet.

Oral Casein Hydrolysates

The nauseating taste of casein hydrolysate was stated by all workers to preclude its routine use as a satisfactory means of treatment of starvation. This difficulty could, of course, be overcome by the use of the nasal tube, but unfortunately the conditions present at Belsen and the lack of staff experienced in gavage prevented a proper trial being undertaken. Furthermore, the physicians at Belsen were convinced that diarrhœa was produced or increased in degree if a strength of 5 per cent or more of hydrolysate was given by mouth or stomach tube. If concentrations of 2·5 per cent or less were used, the total intake of amino-acids which could be given daily was less than that required to establish nitrogenous equilibrium. The report from Holland, however, states that no evidence was obtained that casein hydrolysates of strengths from 5 per cent to 7·5 per cent provoked or increased diarrhœa. This divergence of opinion may be due to the fact that most of the severely ill patients treated at Belsen had grossly inflamed intestinal tracts which may have been particularly sensitive to stimuli of any type.

Comparisons were made in Holland first with varying concentrations and amounts of hydrolysates plus a glucose vitamin mixture, and secondly with a bland diet containing 80 to 300 grams of protein and 2,000 to 3,200 Calories. The conclusion was reached that casein hydrolysates in low concentrations and amounts were totally ineffective. If, however, 2 litres of a 7·5 per cent solution of casein hydrolysate in 10 per cent glucose saline were given daily, good results were obtained, but no better than those achieved by giving a skimmed milk-glucose mixture containing 150 to 200 grams of milk protein. The best results were obtained by giving up to 300 grams of natural protein and 3,200 Calories daily. It may be concluded from the above evidence that oral casein hydrolysates appear to have no place in the treatment of starvation because natural milk protein is equally effective in appropriate quantities, and is cheaper, more readily available and preferred by the patients. Moreover, severe inflammatory diseases of the gastro-intestinal tract, co-existing with starvation, may constitute a contra-indication to oral hydrolysates in concentrations known to be effective.

The Diet in Starvation

Another divergence of opinion is to be noted between the Belsen report and the Dutch report on the question of the quantitative aspects of the initial diet to be prescribed for starving patients. Lipscome lays particular emphasis on the fact that in long-standing cases of severe starvation the body becomes adapted to a low intake of food and water, and accordingly the digestive system will not at first tolerate a large amount of food or rapid increases in diet. For severely ill cases he recommends that the initial diet should contain 800 Calories and 64 grams of protein. The essential constituent of the first diet was 2 litres of milk, either skimmed or reconstituted from dried milk powder, sweetened with 2 oz. sugar.

After three or four days the diet is increased to 1,700 Calories and 92 grams of protein, and finally a few days later to 3,000 Calories and 140 grams of protein. A compound vitamin tablet (containing 1 milligram aneurin, 1 milligram riboflavin, 10 milligrams nicotinamide and 25 milligrams ascorbic acid) was given three times a day throughout the treatment. Burger, Sandstead and Drummond make no mention of the dangers of starting with too big a diet, and recommend an initial intake of 2,500 Calories with at least 80 to 100 grams protein. Patients who were unable to swallow were fed through a nasal tube. The diet should be increased at the earliest opportunity to at least 3,000 Calories and between 200 and 300 grams protein, and for this purpose they recommend the following routine :

7 a.m. .	Porridge made with oatmeal or rice flour and whole, milk, to which three heaped tablespoonfuls of skim-milk powder and 1 tablespoonful of glucose are added. Three slices of bread with jam, meat, cheese, or butter.
10.30 a.m.	'Protein food': 3 tablespoonfuls of skim-milk powder, 1 tablespoonful of glucose, and raw or tinned fruit to improve the flavour (serve cold).
12 noon .	Potatoes, meat, easily digestible sieved vegetables, 'protein food'.
3 p.m. .	'Protein food'.
6 p.m. .	Same as 7 a.m. plus 1 egg.

It is difficult to reconcile the divergences of opinion on the initiation of dietetic treatment in severe starvation except on the assumption that the cases in Holland were neither as severe as the Belsen cases nor as often accompanied by marked inflammation of the gastro-intestinal tract. Under Belsen conditions the authors support Lipscome's views as to the need for proceeding cautiously and by stages, if unfortunate and not infrequently fatal results are to be avoided. The carefully controlled work carried out in Holland, however, clearly indicates the value of a high protein diet which should be prescribed as soon as the patient can tolerat it. Whether an intake of protein as high as 300 grams daily, a recommended by Burger and his co-workers, is in fact necessary o even desirable seems to us a debatable point requiring furth investigation. An intake of 150 to 200 grams of protein daily however, can readily be achieved if the diet contains large quanti ties of a dried skimmed milk powder and glucose mixture and i ingenuity is displayed in the use of flavouring agents and methods of serving the mixture.

The regime that we recommend for the dietetic treatment of a case of severe starvation is as follows :

Days 1 *and* 2. Three and a half pints of skimmed milk (or its equivalent reconstituted from 7 oz. of skimmed milk powder and 3½ pints of water), sweetened with 2 oz. of sugar and flavoured with tea, coffee, or vanilla essence, should be given daily in divided feeds at intervals of 1 to 2 hours, or by gavage if the patient is extremely weak and finds it difficult or impossible to swallow. The following vitamin supplements should be given daily along with these feeds: aneurin 3 to 5 milligrams, riboflavin 5 milligrams, nicotinic acid 50 milligrams, and ascorbic acid 100 milligrams. These supplements should be continued for several weeks. This diet will provide about 70 grams of protein and 1,000 Calories, and not more than 4 pints of fluid.

Days 3 *to* 7. If the patient is responding satisfactorily to treatment the diet should be progressively increased by about 200 Calories per day so as to reach a level of about 2,000 Calories and 100 grams of protein at the end of the first week. This can readily be achieved by the addition to the sweetened milk feeds of the appropriate number of the following dishes, each of which contains about 100 Calories:

1. Scrambled egg, made from one egg.
2. Egg flip made from one egg, teaspoonful sugar, and milk from initial diet.
3. Benger's food or Ovaltine ¾ oz., sugar 1 teaspoonful, plus milk from initial diet.
4. Thin porridge made from ¾ oz. oatmeal, and 1 teaspoonful sugar.
5. Bread 1 oz. and 1 teaspoonful sugar, mixed with milk from initial diet.

By this procedure the recommended Calorie and protein intake is achieved without significantly increasing the fluid intake.

Second Week. If the patient continues to improve, the protein intake should be increased progressively to at least 150 grams and the Calories to 3,000 by the fourteenth day. The diet recommended by Burger, Sandstead and Drummond, quoted on p. 364 fulfils these requirements. Alternatively, the high protein diet No. 29 for the treatment of chronic nephritis with œdema may be used. To increase the protein content up to 200 grams, feeds of the skimmed milk powder-glucose mixture should be given at 10.3 a.m. and in the evening, and the daily milk ration increased t 1 pint. Except in the presence of massive nutritional œdema, th salt restriction ordered in Diet No. 29 need not be imposed. Meat. cheese, potatoes, bread and butter should be given at first in smal helpings, which should be increased in size progressively during th week. The replacement of meat in the diet recommended b Burger by equivalent quantities of fish adds variety, and may b permitted if desired by the patient.

A high protein diet should be continued for several weeks unti the patient's strength, musculature and body weight have bee restored to normal.

For patients *in extremis*, particularly those with vomiting o diarrhœa, parenteral feeding methods will be required at first These have been discussed on pp. 386 to 390.

Experience has shown that apparently mild cases of starvatio may become severely ill within a matter of hours from injudiciou feeding, lack of adequate rest or the development of infection Accordingly we recommend that the initial treatment of starvatio should be the same for both mild and severe cases for the first 4 hours in order to test the patient's powers of assimilation an reaction to the ingestion of food. If, at the end of this period, th clinical state of the patient is satisfactory, the dietetic measure recommended above for the treatment of severe starvation durin the second week can be instituted at once.

CHAPTER XVII

DISEASES OF THE CARDIOVASCULAR SYSTEM

THE action of the heart is influenced by the state of bodily nutrition, and its efficiency is impaired in both of the two opposite conditions, under-nutrition and obesity.

When a person has had to live on a diet grossly inadequate in total caloric value and lacking in protein, 'hunger' œdema, due to a fall in the level of the plasma proteins, may occur, but in addition definite cardiac disturbances are often present. The heart is usually smaller than normal, and the pulse is slow and weak.

It should also be remembered that vitamin B_1 plays an important part in maintaining the efficiency of the cardiovascular system. When the dietary history suggests a vitamin deficiency or when congestive heart failure does not respond adequately to digitalis therapy, concentrated preparations of vitamin B_1 (see p. 46) should be given by mouth or parenterally. Their beneficial effects can be expected within two or three days, the most noticeable feature being an intense diuresis.

Obesity interferes with cardiac efficiency in three ways:

1. The increased body weight due to the deposition of fat leads to a lack of balance between the body mass and the heart's action, and therefore adds to the work of the heart.
2. Fatty changes may occur in the heart, which impair its efficiency. The epicardial fat becomes excessive, and fat may be deposited between the muscle bundles.
3. The accumulation of fat within the abdomen intereferes with the movements of the diaphragm and thus impairs the action of the heart.

The increased body weight and the cardiac embarrassment limit the patient's activities, and as this is usually not accompanied by a lessened food intake, further accumulation of fat completes a vicious circle.

Pathological changes in the myocardium may result from many causes of which the most important are active infections, especially rheumatic fever, sclerosis of the coronary vessels, or long-continued strain of working against a raised peripheral blood pressure. From a therapeutic point of view, heart failure may be divided into three types: (i) congestive heart failure in which the presenting manifestations are dyspnœa, venous distension and peripheral œdema; (ii) dyspnœic failure in which the dominant feature is dyspnœa (cardiac asthma); (iii) anginal failure in which the leading complaint is pain. Evidence of organic disease of the heart may be available for many years before symptoms occur. Such cases of compensated heart disease should be treated essentially by common-sense regulations regarding work and play, so as to enable the individual to live well within the reserve power of his heart. Drugs are usually unnecessary, and *rigid* dietetic restrictions are strongly contra-indicated provided that the patient is not overweight or markedly under-nourished. Sooner or later one or other type of cardiac failure will develop and treatment will then consist of rest, drugs, and the dietetic measures described on p. 369, depending on the severity of the condition.

COMPENSATED MYOCARDIAL DISEASE

Although *rigid* dietary restrictions are unnecessary and indeed contra-indicated because of the harmful psychological effects induced, the following guiding principles should be considered as part of the general reorganization of the patient's method of living. Meals should be regular, well spaced and light. Indigestible foods such as pastries, rich cakes or puddings, preserved or salted meats, or fried foods should be avoided (see p. 265). The evening meal should always be a light one and should be taken early. The patient should be instructed to eat his food slowly and masticate thoroughly. Every meal should be preceded and followed by a rest. Digestive upsets must be guarded against, especially if any degree of chronic venous congestion is present, since the digestion and utilization of the food is impaired by congestion of the gastric mucosa and the liver. Cardiac failure or angina pectoris may be precipitated by over-eating or flatulence. The rules laid down above, that the meals should be light, well spaced, and composed of easily digestible foods, will help to prevent digestive upsets. If flatulence is present, the starchy foods of the diet should be reduced. Condiments, pickles and sauces should be prohibited, and bulky foods containing large amounts of cellulose should be reduced to a minimum.

Fluids should be taken mainly between and not with meals. When there is no œdema, rigid restriction of salt and fluid is unnecessary, and up to 2½ pints of the latter may be allowed daily. Tea and coffee should not be restricted to the point of hardship

If taken weak, these drinks probably are less harmful than is commonly thought, and their diuretic action is useful.

The harmful effects of obesity on the heart have been stressed above. It is obvious that these will act with greater force on a heart already damaged by disease, and therefore it is especially important that cardiac subjects should not become obese. If a patient's activities are curtailed owing to cardiac disease, care must be taken that a corresponding decrease is made in his food intake, otherwise accumulation of fat is very likely to occur. Obesity already present in a patient with heart disease must be treated by the methods given on p. 349, but weight reduction in cardiac cases must be undertaken cautiously, and the weight loss should not be allowed to exceed 1 to 2 lb. per week. Markedly overweight patients should not be brought to or near the ideal weight in one course of treatment. After 14 to 20 lb. have been lost the diet should be slightly increased again, so as to maintain the patient's weight at this new lower level. After a month or two a further reduction in weight can be secured by giving the patient the subcaloric diet again.

CARDIAC FAILURE

The aim of treatment in cardiac failure is to secure the maximum amount of rest for the heart. Since the taking of food of any kind stimulates metabolism, thus causing an increase in the work of the heart, dietary treatment in cardiac failure should be directed towards reducing the amount of food to the minimal amount necessary for the immediate needs of the body.

The diet in congestive cardiac failure should be constructed on the following principles:

1. The caloric value should be low. In the initial stages of the treatment of severe congestive failure, when anorexia is present and nausea is often marked, the diet may with advantage be reduced to about half the basal requirements (*i.e.* about 800 Calories) for a period of a few days. Later, as the patient's condition improves, the diet must be increased, but 1,500 Calories will be sufficient for maintenance purposes as long as the patient is confined to bed or to a chair.

2. Each feed must be small in quantity, easily digestible, and, in the initial treatment of severe cases, fluid or semi-fluid in consistence.

3. The protein quota should be low, since the protein of foods has the greatest effect in stimulating metabolism and therefore increasing the heart's action. The protein quota at first should

be only 30 to 40 grams; with improvement in the clinical condition, this may be increased to 60 to 70 grams.

4. Experimental evidence indicates that glucose has a marked effect in restoring the heart's action in circulatory failure. Therefore the diet should provide ample amounts of easily assimilabl carbohydrate—orange and lemon drinks with sugar, jam, cereals. etc.

5. Since there is accumulation of water in the tissues in congestive failure, the fluid intake must be reduced, and not more tha 1½ pints of fluid as such should be permitted daily. For the sam reason the salt content of the diet should be kept low by usin minimal amounts of salt in cooking, prohibiting its use at table and excluding articles of food rich in salt from the diet (see p. 316)

In the initial treatment of congestive cardiac failure, therefore a diet of about 800 Calories with a fluid intake of not more tha 1 to 1½ pints is advisable. A diet much used on the Continent an in America for such cases is the Karell diet. This consists only o 800 c.c. of milk daily, given in four feeds of 200 c.c. (one smal glassful). No other food or fluid is given. This diet is very low i bulk, carbohydrate and minerals. Its caloric value is only 554 Accordingly it does not fulfil the criteria mentioned above and, i it is employed, it should be only for two or three days, after whic most patients will complain of monotony and thirst.

An alternative to the Karell diet which provides more variet and more carbohydrate without increasing the caloric intake significantly is given in Diet No. 33 (p. 497), and this is the diet whic we recommend for the treatment of severe congestive cardia failure.

When either of these diets is combined with rest and suitabl drug treatment, a rapid disappearance of the œdema and a definit improvement in cardiac efficiency may be expected to occur.

When the patient with severe cardiac failure has progresse towards convalescence, or from the outset in mild cases, a mor liberal diet can be allowed. This should approximate in calori value to the patient's basal requirements (*i.e.* about 1,500 Calories If œdema is still present, the fluid intake should be restricted t 1½ pints, otherwise 2 to 2½ pints may be permitted. Salt restrictio should be maintained by the use of only minimal amounts in cooking and by its prohibition at table. A suitable diet is given in Di No. 34 (p. 497).

After the patient's cardiac decompensation has been relieve as far as possible, the caloric content of the diet can be furth increased to about 1,800 Calories by the addition of three 100-Calorie portions of carbohydrate or fat (see p. 443). This will provide a sufficient diet for the patient's necessarily restricted physical activities. It is advisable to have the patient weighed at regular intervals to ensure that he is not putting on weight.

HIGH BLOOD PRESSURE

In no field of therapeutics has there been less critical judgment displayed and more unnecessary hardship placed on the unfortunate sufferers than in the treatment of high blood pressure. The remarkable variations which may occur in the blood pressure from day to day or even from hour to hour are still not sufficiently appreciated. It is this elusive feature which enables uncritical observers to claim beneficial results from a large number of drugs widely differing in their pharmacological action, or from a variety of diets in which the articles restricted or prohibited may be proteins, fats, carobohydrates, minerals or fluid. Since the cause of essential hypertension is not known, it is wiser to admit that no form of specific thereapy is available and thus, at least, guard ourselves from harming our patients by unwisely restricting too rigidly their activities and their dietetic inclinations. Moderation in everything should be the watchword. When symptoms or signs of heart failure or renal disease develop, the measures outlined on pp. 369 and 326 are indicated. Finally, it is necessary to emphasize once more the great importance of reducing the weight of hypertensive individuals to as near normal as possible. Many of them, but by no means all, are hard workers and over-eaters. The coexistence of disease of the coronary vessels is an additional reason for prescribing an anti-obesity diet (see p. 349). Even one stone in excess of the patient's standard weight is a significant burden on the cardiovascular system and demands correction. The importance of excess weight in producing strain on the heart must be explained to the patient in order to gain his co-operation and to help him to bear the dietetic restrictions which will have to be continued for months. In the writers' opinion it is also advisable to explain, in simple physiological terms, the rational basis on which the diet is constructed.

CHAPTER XVIII

INFECTIOUS DISEASES

METABOLISM IN FEVER

FEBRILE diseases are marked by certain changes in metabolism, which must be taken into account when constructing suitable diets.

1. The Basal Metabolism is Increased

For every degree Fahrenheit rise in temperature, there is a 7 per cent increase in the basal metabolism; this increase is due rather to the infection than to the stimulating influence of the high temperature. As a result of the increased metabolism, there is a greater combustion of nutritive material, which is derived from the body stores and tissues unless a very liberal diet is given.

2. There is a Toxic Destruction of Body Protein

Protein destruction in fever is excessive and may be as much as three times the 'wear and tear' quota of health. Although hyperthermia itself causes some increase in the breakdown of body protein, the protein destruction in fever has little relation to the height of the fever and appears to depend on the severity of the infection.

In a long-continued fever, therefore, an effort must be made to prevent undue loss of body protein by the prescription of a diet in which the protein intake is much in excess of the maintenance requirements in health. There is no ground for the old belief that a high protein intake will increase the height of the fever, for it has been shown that protein does not exert its usual specific dynamic action in the presence of fever.

Apart from these theoretical considerations, the arrangement of the diet is made difficult by the anorexia which is present in most fevers, and which may be complicated by nausea and vomiting. It may be mentioned here that much can be done to combat the anorexia by ensuring that the mouth is kept clean. If the tongue is furred and sordes are allowed to accumulate, it is not surprising that the patient's distaste for food will be enhanced. The mouth should be washed or swabbed out with a weak solution of sodium bicarbonate or other mouth-wash before and after every meal.

THE DIET IN FEVER

It is best to consider this under separate headings – long-continued fevers, *e.g.* enteric fever; fevers of short duration, *e.g.* pneumonia; and chronic infections, *e.g.* tuberculosis.

1. Long-continued Fever

The principles governing the construction of the diet in these circumstances are:

1. The diet must have a high caloric value – up to 50 per cent more than the normal maintenance intake. This means that the diet should yield about 3,000 Calories.

2. A liberal protein quota must be provided, in an endeavour to make good the excessive destruction of body protein. Owing to the anorexia and the necessity for providing food in liquid or semi-solid form, it may not be possible to give more than 70 to 90 grams of protein daily, but this intake will usually cover the protein loss except in the most severe fevers.

3. A large proportion of the food ingested should be in the form of carbohydrate. This will prevent ketosis, which may arise if the patient is burning his own body fat in increased amounts owing to the raised metabolism. It will also replenish the liver's store of glycogen, which in fever is burned up before body fat is drawn upon. A liver rich in glycogen is more resistant to toxins than one poor in glycogen. Lastly, the 'protein sparing' property of a high carbohydrate diet is of practical importance.

4. Food should be served in liquid or semi-solid form, and must be easily digestible. Large meals should be avoided, and the principle of small feeds every 2 to 3 hours adopted.

5. Fluids must be given in abundance. Owing to the loss of fluid in sweat and the disinclination of patients to take fluids and food, a negative water balance may develop in fever. Fluids require to be given in greater amounts than are usually ingested, but excessive amounts must not be forced on the patient. A daily total fluid intake of about 5 to 6 pints (3,000 to 3,600 c.c.) is ample.

Salt depletion occurs from sweating and this must be made good by adding sodium chloride to suitable feeds and to the glucose lemonade drinks (see Diet sheet No. 35). The strength recommended, ¼ teaspoonful to the pint, gives approximately ¼-normal saline, the taste of which will be adequately disguised by fruit juice and lactose.

The Construction of the Diet. Milk is the mainstay of the diet in fever, and should be given in large amounts, say 1½ to 2 pints daily. It may be citrated, peptonized or diluted with water. Apart from its valuable nutritive qualities, the fluid form and relative lack of taste of milk make it a most suitable food for febrile patients It is useful, also, in providing a vehicle for the administration of other foods, such as eggs, cream and sugar.

Protein. About half the desired protein intake will be obtained from the milk (40 grams in 2 pints). The remainder is best given in the form of eggs (which can be boiled, poached, scrambled, or switched in milk or fruit juice), or cream cheese (unless the diet is completely fluid). There is no objection to giving small helpings of minced chicken or pounded white fish, provided that the patient can take these without suffering any gastro-intestinal upset.

Carbohydrate. Foods rich in starch should be given, but thos with a large cellulose content should be omitted. Cooked cereal such as Benger's or Allenbury's Foods, or Cream of Wheat serve with cream and sweetened to taste are both appreciated and easil digested by febrile patients. Farex (Glaxo) may be specially mentioned in this connection as it comprises a cereal food to whic minerals and vitamins have been added. Toast or rusks sprea with honey or jelly are allowed. Fruit juices should be given i liberal amounts to provide an ample intake of vitamin C, th requirement for which is increased in fever; they are also useful a flavouring agents for the increased amount of water which shoul be given. Fruit drinks sweetened with sugar can also be recommended as valuable sources of Calories. Lactose, being much le sweet than cane sugar or glucose, can be given in larger amount ½ to 1 oz. will not make 5 oz. of orange juice unduly sweet, but ca must be taken not to produce diarrhœa through the excessi administration of lactose.

Fat is given in the form of cream, butter, vitaminized margari and egg yolk. As much as ½ pint of 20 per cent cream may taken daily.

The diet shown on page 498 (Diet No. 35) is a completely flu one, which may be used in the early stages of a long-continu fever such as typhoid. It contains approximately 3,000 Calorie

As the patient improves and can take solid food, this diet shou be modified progressively by the addition of toast or bread wi butter or margarine, milk puddings, custards and stewed or fr fruit, puréed vegetables, and cream of chicken, rabbit and fish, a some of the feeds of the fluid diet can then be omitted.

In Convalescence, the appetite is usually increased, but it is b to proceed slowly and not allow a food intake too great for the patient's power of digestion. The food should be easily digestible and rich in protein and vitamins. Foods such as pastries and cakes, and highly seasoned and indigestible articles (see p. 265) should be avoided. A suitable diet for use during convalescence is given in Diet No. 36 (p. 499).

2. Short Febrile Illnesses

In fevers of short duration such as pneumonia, in which the result, favourable or otherwise, is a matter of days not weeks, the diet should be constructed with the chief object of saving the patient from all possible exertion in the taking of his food; the actual amount of nourishment given is of less importance.

The diet should be an entirely fluid one, and should be given in the form of small feeds at frequent intervals, usually every 2 to 3 hours. Milk is again the mainstay of the diet, and 2 pints should be given daily. If necessary, it may be citrated (1 grain of sodium citrate to 1 oz. of milk) or peptonized, and some of the milk feeds should be flavoured with cocoa, coffee or tea, or given as an egg switch. Two or three eggs may be given daily, switched in milk, or as the egg and fruit drink given in the fever diet on p. 498. Liquid cooked cereals such as a gruel or any of the proprietary preparations, served with cream, and cup custards may be given at some of the feeds to vary the diet. Fruit juice should be given in ample amounts for its vitamin content and also as a flavouring agent for the water, which should be given to the patient between feeds in liberal amounts.

One of the distressing complications of an acute febrile illness such as pneumonia is tympanites. Since the principal factor in its causation is a toxic paralysis of the bowel, correction of the toxæmia by appropriate means should be the basis of treatment, *e.g.* serum, sulphonamide, the intravenous administration of glucose, saline or blood, etc. Nevertheless, measures for the relief of symptoms may be of great value. These include drugs, such as pituitrin and doryl, and dietetic restrictions. If the motions are loose and contain excessive amounts of curd, cream should be prohibited and milk reduced in quantity and citrated or diluted with water. The caloric intake may be maintained by fruit juices sweetened with glucose or lactose. On the other hand, if there is no evidence of impaired fat digestion and absorption, the tympanites should be treated by restriction of carbohydrate foods, particularly those rich in cellulose and starch.

The use of alcohol in fever has already been dealt with (see p. 198) It has for long been regarded as a food and a stimulant in pneumonia. Opinion at the present time regards alcohol as a sedative which is valuable in the treatment of elderly febrile patients, bu which need not be given to younger persons who are not accustomed to it. It is widely held that alcohol should be given durin the course of a fever to persons who have been accustomed to it use in excessive amounts, as addicts are liable to develop deliriu tremens during febrile illnesses.

3. Chronic Infections

Tuberculosis, excluding the acute forms such as miliary tuberculosis, is the most common example of a chronic infection i which careful arrangement of the diet will do much to maintain th patient's general resistance. Tuberculosis is a debilitating diseas and emaciation is a prominent feature. While an attempt must b made to combat this emaciation by a liberal diet, it is necessary t give a warning here against forced feeding with the idea of gettin the patient to put on as much weight as possible, even to the poi of obesity. This is an error in treatment, and may actually prov harmful in the long run. The aim of dietetic treatment should b to raise the tuberculous patient's body weight to the calculate ideal or a few pounds over it, and not to make him fat.

In constructing the diet, it should be remembered that a wel balanced diet is even more necessary in a tuberculous patient tha in a healthy individual. Therefore it is not sufficient to prescri merely milk, cream, and eggs in large amounts, and leave the re of the diet to the patient's discretion. The diet should be amp in Calories – slightly more than the patient's calculated mainte ance requirement. Thus for a man of 10 stone who is ambula but not doing physical work, an intake of 2,500 to 3,000 Calori is sufficient. Toxic destruction of the body protein is less in chron tuberculosis than in the acute fevers, so that a protein quota of 1 to 110 grams (*i.e.* just over the amount usually taken by a healt person) is adequate. The diet should be rich in fat; this ensures good intake of the fat-soluble vitamins, and fat ingestion, unli that of carbohydrate, causes but little increase in respirato activity. Digestive disturbances call for rearrangement of the di They are commonly due to attempts to over-feed emaciate febrile patients whose digestive powers are depressed. For instan the forcing of excessive quantities of fatty or starchy foods m lead to diarrhœa or flatulence respectively.

While a rigid dietary prescription is unnecessary, Diet No. 37 (p. 500) can be used as a guide. To ensure a liberal intake of vitamins, cod-liver oil or one of the concentrates (adexolin or radiostoleum) should be prescribed to provide vitamins A and D, while vitamin B can be supplied as Bemax, yeast tablets (two thrice daily) or Marmite, or yeast (see pp. 47 to 50).

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CHAPTER XIX

ANÆMIA

THERE are two main types of anæmia for which dietetic treatment is of value: (i) iron deficiency anæmia (hypochromic microcytic anæmia) whether due to dietary deficiency, acute or chronic hæmorrhage, or sepsis and toxæmia; (ii) megalocytic anæmia (hyperchromic macrocytic anæmia) as is present in Addisonian pernicious anæmia, tropical sprue, idiopathia steatorrhœa, etc.

It is necessary to draw attention to the need for accurately establishing the type of anæmia present and, in the case of hypochromic anæmias particularly, for making a most careful search for the causal factor. The failure to do so may result not only in an ineffective response to treatment but may allow serious organic disease, such as cancer, to progress to an inoperable stage. It is very desirable to point out that once anæmia is established it is both unwise and uneconomical to rely solely on diet for its correction, since this object can be much more effectively obtained by the oral administration of medicinal iron in hypochromic microcytic anæmia and by the parenteral injection of liver extract in megalocytic anæmia. On the other hand, for the prevention of anæmia and the relapses which tend to follow the established condition, the prescription of a diet rich in the principles required for the construction of the stroma and hæmoglobin of erythrocytes is of the greatest value.

ANÆMIA DUE TO IRON DEFICIENCY

Chronic nutritional hypochromic anæmia is a disease which occur frequently among women and infants of the working classes an leads to much ill-health and inefficiency. Its cause lies essentiall in the failure of the diet to meet the nutritional requirements fo iron (see p. 141). For a fuller consideration of this problem th reader is referred to the paper by Davidson and Fullerton (193 and 1942).

Treatment. In all types of anæmia when the blood level falls t 000 red cells or less, anorexia, nausea, vomiting and oth intestinal symptoms tend to occur. No attempt should b this stage to give large quantities of meat, liver and gree vegetables, as the stomach cannot tolerate them. A bland, easily digested diet is recommended, divided into small meals at two to two-and-a-half hourly intervals. The basis of the diet should be milk and milky foods, jellies and custards, lightly boiled or scrambled eggs, pounded fish, purées of fruit and vegetables and small amounts of thin bread and butter. Patients in the severely anæmic stage with marked gastric disturbances require hospitalization for nursing and dietetic reasons. With the rise of the hæmoglobin level, appetite and digestion improve, and the diet should be cautiously increased by the addition of chicken, meat and liver, vegetables and raw fruit. The aim in treatment of the iron deficiency anæmias is to reach a diet containing at least 15 milligrams of iron a day. Before the introduction of the National wheatmeal loaf the economic conditions of the poor were such as to make the purchase of a diet containing this quantity of iron difficult or impossible. Should it be found that the normal requirements for iron conditioned by pregnancy or menstruation cannot be satisfied by a maintenance diet containing approximately 15 milligrams of iron a day, the administration of medicinal iron for one week in each month must be advised after the normal hæmoglobin level has been restored.

No attempt should be made to bring the hæmoglobin level to normal by dietetic measures alone, since we have shown that this
is both uneconomic and unsatisfactory. This does not mean th
we should be content with low iron diets (5 to 10 milligrams dai
since a figure of at least 15 milligrams daily can be attained chea
and easily by a proper selection of foods. In addition, an impro
ment in the protein, mineral and vitamin content will res
Accordingly, Table 12 (p. 381) is submitted in order to indicate
articles of diet most suitable for this purpose.

Prophylaxis

For the prevention of chronic nutritional hypochromic a
diet containing ample quantities of the foods rich in ir
greatest value. At the same time an adequate supply
taining vitamins B_1 and C should be included in the
former is of value in maintaining the normal
alimentary canal including absorption, while the l
for the normal formation of the erythrocyte. Th
these vitamins are discussed on pages 46

In Aberdeen it was found that man
assistance were able to spend only 6d.

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diet shown on Table 11, constructed in 1937, yields 2,100 Calories, 70 grams of protein and 10 to 14 milligrams of iron at a cost of approximately 7d. If National wheatmeal bread had been eaten instead of white bread the daily intake of iron would have been

TABLE 11

ECONOMICAL DIET WITH SATISFACTORY IRON CONTENT

	Iron Content mg.	Cost in Pence
Breakfast		
Porridge (1½ oz. oatmeal)	1·8	0·16
2 oz. white bread	0·4	0·31
Dinner		
2 oz. meat, cooked	3·4	1·00
3 oz. potato, boiled	0·3	0·10
3 oz. cabbage, boiled	0·3	0·25
Milk pudding, (made from ½ oz. cereal)	0·1	0·10
2 oz. white bread	0·4	0·31
Tea		
2 oz. oatcakes	2·4	0·22
Supper		
1 herring fried in oatmeal (3 oz. flesh)	1·5	1·00
1 oz. white bread	0·2	0·15
1 oz. oatcakes	1·2	0·11
Rations per Day		
10 oz. milk	0·3	1·50
2 oz. vitaminized margarine . . .	—	0·90
1 oz. syrup	0·4	0·22
1 oz. sugar	—	0·16
½ apple or ½ orange.	0·2	0·50
Total . . .	12·9	6·99

tives to Meat or Fish Dishes	Iron	Cost
entil soup (2 oz. lentils per person)	4·4	0·50
usage, beef, fried (3 oz.) . . .	3·3	1·13
se (2 oz.)	0·4	0·50
. . . .	1·2	1·00

Total Calories, 2,100. Protein, 70 grams.
approximately 7d.; compiled 1937.

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TABLE 12

IRON CONTENT OF COMMONER FOODSTUFFS

Foodstuff	Total Iron mg. per ounce	Cost per pound	Mg. of Iron for 1d.
1. *Flesh Foods*			
Liver, ox, raw	3·9	1/– to 1/2	4·8
Sausage, beef, fried	1·1	4d. to 8d.	2·9
Beef, raw	1·1	6d. to 10d.	2·2
Corned beef, tinned	3·1	7d. to 10d.	5·8
Rabbit, stewed	0·5	2½d. to 5d.	2·1
Herring, fried	0·5	1d. each	1·8
White fish, steamed	0·2	8d. to 10d.	0·3
Egg	0·8	1d. to 1¼d. each	1·3
Cheese	0·2	6d. to 8d.	0·5
2. *Cereals, etc.*			
Oatmeal	1·2	1½d. to 2d.	11·0
National bread, 85 per cent extraction (1942–4)	0·5	2¾d.	2·9
National bread, 80 per cent extraction (1945)	0·3	2¾d.	1·7
White bread, 70 per cent extraction	0·2	2½d.	1·3
Rice, polished	0·1	2d. to 3d.	0·6
Golden syrup	0·4	3d. to 4d.	1·8
3. *Vegetable Foods*			
Lentils, raw	2·2	2d. to 2½d.	15·6
Split peas, raw	1·3	2½d. to 3½d.	6·9
Cabbage, raw	0·3	1d. to 1½d.	3·8
Leeks, raw	0·4	1½d. to 2d.	3·7
Potato, raw	0·2	½d. to ¾d.	5·1
Carrot, raw	0·2	2d. to 3d.	1·3
Onion, raw	0·1	2d.	0·8
Turnip, raw	0·1	¼d. to ½d.	4·2
Apple, raw	0·1	1d. each	0·3
Orange juice, raw	0·1	1d. each	0·2

increased by 1·5 milligrams daily. It is not suggested that the diet contains optimal amounts of other minerals and vitamins, but it was the best procurable for the prevention of anæmia when such small amounts of money were available. For those who are financially better off, the addition of the flesh foods shown in Table 12 will make the diet not merely more pleasant but more efficient in its blood-forming properties. Additional quantities of fresh fruit and vegetables should be ordered. A study of Table 12 shows that the flesh foods, eggs, oatmeal, wheatmeal flour, lentils and split peas are articles of food particularly rich in iron. It does not follow, however, that the efficacy of any foodstuff in promoting hæmoglobin regeneration depends solely on its iron content. The availability of the contained iron, which in turn depends on the ease with which ionizable iron is liberated by the processes of digestion, may be a factor of importance. Recent work suggests, however, that undue emphasis has been given to this aspect in the past (see p. 139). In any case, it has been found that, with the exception of beef and mutton, the foods richest in iron (over 2 milligrams per 100 grams) have also the highest content of available iron, and accordingly all that the family doctor need know is the foods of a high total iron content included in Table 12 (p. 381) – *i.e.* liver, sausage, eggs, oatmeal, lentils, split peas and wholemeal bread.

As the economic factor is of such importance, we have shown in Table 12, column 4, the amount of iron in milligrams which can be purchased for one penny, and we draw particular attention to the cheapness of 'food iron' in oatmeal, lentils and split peas. The costs of the different foods (with the exception of National bread on which the figures in column 3 are based, were obtained in 193 directly from the shops used by the poorest classes in Aberdeen.

PERNICIOUS ANÆMIA AND OTHER MACROCYTIC ANÆMIAS

The aim of treatment is (i) to restore the blood picture, qualitatively and quantitatively, to normal as quickly as possible; (ii) to maintain a normal blood level; (iii) to replenish and stock adequately the depots of the body with the factors necessary fo blood formation. Minot has rightly pointed out that the individua who receives only just enough of a nutritional factor, such as vitamin or a mineral, to maintain health, may be precipitated int the zone of partial deficiency with the advent of infection; henc the importance of adequate reserves.

It would appear advisable to consider the treatment of pernicious anæmia according to the stage of the disease present whe the patient is first seen.

The Severe Relapse Stage. The patient is critically ill and in collapsed state, with a blood count of approximately 1 million re cells and a hæmoglobin content of 20 to 30 per cent. Treatmen consists of rest in bed, parenteral injection of liver extract, tran fusion of blood if the clinical condition suggests that the patient may not survive the few days which must elapse before the effects of the liver extract can appear, and the giving of a bland diet as already described on p. 379.

Stage of Moderate Relapse. The patient complains of weakness, palpitation, dyspepsia, and dyspnœa on effort, but there is no immediate danger to life. The blood count may be from 1½ to 2½ million red cells and the hæmoglobin 40 to 60 per cent. In such a case a choice must be made between treatment with liver extract given parenterally, or liver, liver extract or a preparation of hog's stomach given by mouth. In the authors' opinion parenteral treatment is in general the most efficient and economical method available. By its use progress is so rapid that no special diet is required. The patient should take a plentiful, well-balanced, mixed diet containing meat, liver or kidney and green vegetables at least at one meal a day.

Maintenance Treatment is best carried out for life by giving an injection of a potent liver extract at intervals of 2 to 6 weeks, depending on the dose and preparation employed and the individual patient's response. No special dietetic instructions are required other than those mentioned in the previous paragraph. It is of considerable interest to note that, provided a satisfactory blood level is maintained, no gastro-intestinal symptoms are present in the vast majority of cases, despite the fact that apermanent achylia gastrica exists.

ANÆMIA IN SPRUE (TROPICAL AND NON-TROPICAL)

The dietary regime for the treatment of the steatorrhœa of sprue has been described on p. 285. Anæmia develops in all the more severely ill patients. It may be either of the iron deficiency type or of a megalocytic type due to a deficiency of the specific anti-anæmic factor. In the former medicinal iron and in the latter liver extract should be given. In some cases of sprue with a macrocytic hyperchromic blood picture, the response to parenteral liver therapy is poor as compared with that obtained in cases of classical Addisonian pernicious anæmia. Proteolysed liver given by mouth may produce a dramatic response (Davis and Davidson, 1944). In addition a high protein diet should be prescribed. A figure of at least 150 grams daily may be required. This may be secured by giving frequent feeds of sprulac (see p. 285), liver and liver soup, and underdone beef steak from which all visible fat has been removed.

The remarkable clinical and hæmotological effects of folic acid recently reported by Spies (1946) in the treatment of sprue and other macrocytic anæmias is discussed on page 67. The benefits of the high protein diet mentioned above may be due to its high content of folic acid.

CHAPTER XX

SPECIAL FEEDING METHODS

THE administration of fluid, salts and food by the natural channels may be undesirable or impossible under certain circumstances. Thus, the surgeon may wish to keep the stomach completely empty following major surgical procedures: the entrance of food into the stomach may be prevented by inflammation, malignant disease or mechanical obstruction in the mouth, pharynx or œsophagus: weakness or paralysis of the muscles of deglutition secondary to disease of the brain or peripheral nerves may make the act of swallowing difficult or impossible. Even when no dysfunction of the mouth, pharynx or œsophagus is present, special feeding methods may be required if vomiting is persistent and severe or if profuse diarrhœa hastens food and fluid too rapidly through the intestines. Likewise if coma, delirium or insanity is present, ordinary feeding methods may be difficult or impossible. Marked malnutrition and dehydration may result from long-continued severe toxic and infective states, extensive burns and major surgical operations, and their correction may require that the oral administration of food and fluid be supplemented in part or in whole by special feeding methods.

It is necessary to emphasize that such methods should be considered as emergency procedures to tide the patient through a crisis. Technical difficulties and the discomfort of the patient limit their time of application to a matter of days or at most weeks. While they are undoubtedly life-saving procedures in specially selected cases, they must not be considered as adequate substitutes for the ingestion of fluid and food by mouth.

Special feeding methods can be broadly divided into two classes according to the route by which the nutrients are introduced into the body:

1. Parenteral, *e.g.* by subcutaneous, intramuscular or intravenous infusion.
2. Enteral, *e.g.* by the introduction of a tube into the stomach, duodenum or jejunum via the mouth and œsophagus, through a gastrostomy opening, or into the rectum.

One important difference between parenteral and enteral feeding methods is that until the recent introduction of casein hydrolysates (see below) the former were capable only of maintaining the patient in fluid and salt balance and of supplying some energ in the form of carbohydrate. Experience has shown that th parenteral administration of fat and protein was not only ineffective but actually dangerous. On the other hand, all th nutrients required for the maintenance of health and strength ca be given provided a tube can be inserted into the stomach.

PARENTERAL SPECIAL FEEDING METHODS

1. Subcutaneous Administration

As has been noted above, fat and protein should not be administered by any parenteral route. Glucose is absorbed slowly fro the subcutaneous tissues, but its use by this route is not recom mended because of its tendency to cause irritation and facilitat infection. It follows therefore that only fluid and salt should b given by the subcutaneous route, and accordingly it is obvious th the patient's energy and nitrogen requirements will be unsatisfie The method is, however, of real value in the treatment of dehydr tion and hypochloræmia of moderate degree in patients who a unable to take or retain adequate quantities of fluid by mout Up to 1,500 c.c. of normal saline (0·9 per cent) can be given i twenty-four hours. If the patient is taking a certain amount fluid by mouth, this quantity may be sufficient to restore the flu balance, while the salt content is in excess of the daily intake a normal individual.

Technique

The sites of election for subcutaneous administration of fluid a (1) the outer border of the pectoralis major muscle, and (2) t anterior internal aspect of the thigh. Special 'subcutaneou needles of medium bore and with lateral apertures should employed. They are inserted through the skin in the direction the apex of the axilla and Poupart's ligament respectively. Bo sides of the patient are used simultaneously, the two needles wi suitable adaptors being connected by a Y-tube to the delive tube attached to the drip apparatus. The saline container shou be secured about 2 feet above the level of the patient. All t apparatus should be carefully sterilized, and after insertion t needles should be covered with a sterile dressing and secured wi adhesive strapping. If 5 c.c. of 2 per cent procain is added to ea 500 c.c. of saline, the patient will suffer little discomfort. The ra of flow should be adjusted to approximately 40 drops a minute, and if absorption is slow, as evidenced by the presence of undue subcutaneous œdema, the flow should be stopped temporarily. The needles should be withdrawn and reinserted in another situation every forty-eight hours.

2. Intramuscular Administration

What has been said in the previous paragraph about the inadvisability of giving fat, protein and glucose by the subcutaneous route is equally applicable to intramuscular administration. Fluid and salt are more quickly absorbed from muscles than from subcutaneous tissues, but this advantage is more than counterbalanced by the relatively small amount of fluid that can be given without producing tension in the muscle with consequent pain to the patient. Accordingly, the intramuscular route cannot be advised when considerable amounts of fluid are required for the correction of dehydration.

3. Intravenous Administration

The technique of infusion through a needle or cannula inserted into a vein is sufficiently well known as not to require description here.* Attention, however, must be directed to the great importance of using only fluids which are especially prepared for intravenous infusion if reactions are to be avoided. Fluid may be given as a single infusion to be repeated at intervals as required, or it may be given continuously by the drip method. Single infusions usually comprise some 500 c.c. of fluid, and may be repeated two, three or four times in the 24 hours. The time recommended for the infusion of each half-litre is approximately three-quarters of an hour. If severe shock is present it may be necessary to give the first half-litre in fifteen minutes. When the continuous drip method is employed, from 2 to 3 litres of fluid may be given in 24 hours. A careful watch on the heart for signs of dilatation, and on the lungs and subcutaneous tissues for evidence of œdema, must be undertaken when large amounts of fluid are given intravenously, especially to severely ill patients. A record of the intake and output of fluid must be carefully kept.

Fat and protein must never be given intravenously. Until recently water, salt and glucose have been the only nutrients which could be safely administered. When hypochloræmia and dehydration are both present, it should be realized that the amount of fluid required to correct the dehydration, if given in the form of normal saline, may contain sodium chloride considerably in excess of the patient's needs, thus leading to a waterlogging of the tissues. It is dangerous to administer more than 1,500 c.c. of normal saline in 24 hours for more than one or two days unless laboratory facilities are available for the repeated determination of the chloride content of the blood. The remaining 1,000 to 2,000 c.c. of fluid required to correct the dehydration should be given in the form of a 5 or 10 per cent solution of glucose in sterile water. Glucose is of value in abolishing ketosis and in supplying energy. Its value in the latter respect, however, must not be exaggerated, since an infusion of 3 litres of 10 per cent glucose solution would supply only 300 grams of sugar, a quantity which barely suffices to meet the basal energy requirement, while the patient's nitrogen needs would be practically unaffected. The concept that intravenous infusions of glucose saline can by themselves restore a patient's loss of weight and strength is quite erroneous. On the contrary, wasting of muscle and weakening of the patient will continue progressively. Accordingly, it may be said that as a means of overcoming a temporary crisis the infusion of glucose saline is of inestimable value. But unless the patient recovers his powers of ingesting adequate amounts of all essential nutrients, or unless these nutrients can be transferred to his stomach through a tube, inanition followed by death is bound to occur. The whole position, however, has been entirely altered by the discovery that mixtures of pure amino-acids or specially prepared solutions of casein hydrolysates can be given intravenously, with the result that nitrogenous equilibrium can be restored and maintained over periods lasting several weeks. Of such practical importance is this discovery that it is proposed to devote a special section to its discussion.

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* Readers who wish to refresh their memory are referred to the section on technical procedures in the *Textbook of Medical Treatment* by Dunlop, Davidson and McNee, 4th Ed., p. 863.

Intravenous Alimentation with Amino-Acids

In the past few years it has been repeatedly demonstrated both in animals and in human beings that the administration, orally or intravenously, of a suitable mixture of amino-acids, in the pure state or obtained from the hydrolysis of protein with acids or enzymes, will maintain nitrogenous equilibrium and regeneration of plasma protein. The disadvantage of acid hydrolysis is that destruction of certain amino-acids, notably tryptophane, may result, and unless the digest is supplemented with the missing amino-acids, nitrogenous equilibrium will not be established. This disadvantage does not occur with enzyme digests and accordingly this method has been widely used to break down casein into its constituent amino-acids. The enzymes most frequently used are trypsin and papain. (See Gaunt (1944) for a review of the literature on the intravenous administration of protein hydrolysates.)

The preparation of protein hydrolysates has now passed from the experimental to the commercial stage. A hydrolysate of casein especially prepared for both oral and parenteral use is marketed under the trade name of 'Amigen' by Mead, Johnson & Co., U.S.A. Casein hydrolysates prepared by British manufacturing firms are also now available. All the essential amino-acids are present in Amigen including methionine in a concentration of 3 per cent; 65 per cent of the total nitrogen is in the form of amino-nitrogen. Amigen is a powder readily soluble in water. It can be given in 5 to 10 per cent solution with an equal amount of glucose by intravenous infusion or by mouth, or better still by stomach tube because of its unpleasant taste. Glucose should always be given with protein hydrolysates in order to prevent the amino-acids being used as a source of energy. The dose of Amigen recommended for protein-depleted patients, either orally or intravenously, is 1·5 – 3 grams per kilogram body weight daily.

Intravenous administration of protein hydrolysates is advised when oral feeding is contra-indicated, difficult or ineffective. It has been used in the treatment of the following conditions: Obstruction of the alimentary tract; persistent vomiting; pre-operatively in undernourished patients, and post-operatively as a supportive measure. The administration has also been recommended where the assimilation of protein is defective – *e.g.* intractable diarrhœa, ulcerative colitis, and various types of steatorrhœa, particularly pancreatic fibrosis. Lastly, casein hydrolysates have been found very effective as a means of accelerating the regeneration of protein lost from extensive burns and from hæmorrhage. It should be noted, however, that for the initial treatment of shock, plasma should be given, because protein hydrolysates have not the same stable osmotic effects. Undesirable reactions (flushing, headache, vomiting, rigors, fevers) resulted from the intravenous infusion of some of the original preparations. These have now been largely eliminated in the American preparation 'Amigen' as a result of improved manufacturing technique. Infusion should always be given slowly, as by this means reactions occur much less readily and loss of amino-acids by renal excretion is reduce to a minimum. The speed of infusion of 5 to 10 per cent case hydrolysate solutions should not exceed 200 c.c. per hour. Man clinical records have now been published which prove conclusivel that it is possible to give up to 200 grams of casein hydrolysa daily by the intravenous route to patients for several weeks, ar that during this period body weight will be maintained or improve and a positive nitrogen balance secured.

Nevertheless we feel that there has been a tendency to exagge ate the value of this new method of alimentation, and to minimi the dangers of venous thrombosis and reactions. Our views a perhaps coloured by the experiences related on pp. 358 to 3 concerning the treatment of severe malnutrition from starvatio which showed that even critically ill patients are able not mere to swallow a skim milk-glucose mixture, but to digest and abso it as well. We feel therefore that intravenous alimentation wi amino-acids should be reserved for the treatment of the conditio mentioned above where these are leading to grossly defecti assimilation of protein.

ENTERAL SPECIAL FEEDING METHODS

1. Gavage

The term gavage is used to describe the procedure of supplyi g nutrients to a patient through a tube introduced into the stoma via the mouth or nose.

Indications

Gavage is indicated in patients who are unable to eat because (1) inflammation of the mouth, pharynx or œsophagus; (2) p alysis or paresis of the muscles of deglutition; (3) nausea and loss appetite. Vomiting is not necessarily a contra-indication to gava since patients may retain food given by the gastric tube whi would be immediately returned if taken by mouth; (4) deliriu coma and insanity causing the patient to refuse food. It is a indicated in severe toxic and infective states and after ma operations, excluding the surgery of the stomach, when the patie is grossly debilitated and dehydrated, and is unable to ta sufficient nourishment by the normal channels to build up strength; and as a therapeutic measure for peptic ulcers which to respond to treatment by the orthodox methods described Chapter XII.

ontra-indications

Gavage is contra-indicated in patients who have recently under- one operations on the stomach. It is obviously impracticable if ere is an obstruction to the passage of the tube in any situation tween the mouth and the stomach, or in the presence of pyloric enosis or high intestinal obstruction.

dvantages of Gavage

Gastric feeding through a tube has many advantages over arenteral and rectal feeding (see below).

(1) Since the nutrients introduced into the stomach reach the tissues through the normal channels, the dangers associated with parenteral feeding (sepsis, cardiac dilatation, pulmonary œdema, etc.) are largely obviated.

(2) All types of nutrients in adequate amounts can be supplied, including inorganic salts and vitamins.

(3) Foods and medicines which may be distasteful to the patient can be given without any distress.

(4) The total quantity of fluid which can be given daily by the gastric tube is at least twice the amount which can safely be introduced parenterally. This is a matter of great importance in dangerously dehydrated patients such as are encountered not infrequently in the tropics.

(5) Gavage may be continued for weeks on end without danger or real discomfort to the patient if the trans-nasal route is used. Its only rival, intravenous infusion of glucose and amino-acids, presents many technical difficulties if it has to be carried on for more than a few days.

(6) If it is desired at any time to empty the stomach, its contents can be aspirated through the tube, and this can be followed by gastric lavage if deemed necessary.

onclusions

From what has been said, it is apparent that, in suitable cases, vage is the method of choice when special feeding methods are dicated. So satisfactory have we found the method of feeding rough the trans-nasal route, that we are surprised that it is not ore commonly used. Accordingly we feel it is advisable to escribe in some detail the technique employed.

echnique

It was formerly the practice to pass a wide-bore stomach tube hrough the mouth into the stomach. The nutrients were poured into a funnel attached to the tube, and thereafter the tube was removed. The procedure was repeated three or four times in the 24 hours. Far more satisfactory results are obtained if a thin soft rubber tube is passed into the stomach via the nose. While a Ryle's tube will be found suitable for the introduction of glucose saline or milk, a soft rubber tube with lateral openings at its end (nasal catheter size No. 9) should be used for running in the less fluid pabulum described below. Patients find the soft nasal catheter quite as comfortable as the Ryle's tube. The distance from the nostril to the stomach varies from 22 to 26 in. in different individuals. The tube should be lubricated with olive oil or liquid paraffin. While usually unnecessary, in nervous patients the nose may be sprayed with a solution of procaine. It will generally be found that one nostril is more patent than the other, and this side should be used for the initial passage of a tube. When about 8 in. of the tube have been inserted it will have reached the pharynx, and the patient will tend to cough or retch. At this point he should be encouraged to swallow with the result that the tube will enter the œsophagus. Thereafter it can be pushed quickly down into the stomach with little or no discomfort to the patient. Little difficulty will be found in patients who co-operate and who retain the swallowing reflex. When this reflex is lost, or when a patient is in coma, or delirium, the tube may have to be directed manually behind the epiglottis into the entrance of the œsophagus. The insertion of a thin wire stylet inside the tube may facilitate its passage. It is absolutely essential in all cases to be sure that the tube has passed into the œsophagus, and not into the trachea, since the introduction of fluid into the lungs causes intense discomfort to the patient, and may even lead to a fatal result. If the tube is in the trachea or bronchus, dyspnœa and coughing occur immediately, and air can be felt and heard issuing from the outlet of the tube.

The most important point to remember is that fluid should never be introduced into the tube unless a sample of gastric juice has first been aspirated. Having made certain that the tube is in the stomach, it is strapped to the forehead and connected with a gravity drip apparatus, fixed in position to a stand about two feet above the level of the patient. The rate of flow is controlled by a screw clamp. Delirious and insane patients may attempt to pull the tube from the nose, and this must be prevented by adequate sedation and, if necessary, by splinting the arms. Although we have left a tube in position in the same nostril for six days without any apparent harm, we recommend that the tube should be withdrawn every 48 hours, washed and boiled, and reinserted into the stomach through the other nostril. Once in position, the patient complains of little or no discomfort, and no interference with sleep usually results. Another advantage of the trans-nasal route is that it leaves the mouth unobstructed so that the patient can cough and spit in comfort, and can supplement the gastric feeds by oral ingestion if he is able and wishes to do so.

Nutrients

Provided the nutrients are in a sufficiently fluid state to pass down the tube, there is no contra-indication to any particular food. Nutrients may be given by continuous drip or intermittently every two or three hours. The former method is the one we recommend. In the treatment of resistant cases of peptic ulcer, we have shown, by testing samples withdrawn hourly for 24 hours, that a continuous state of achlorhydria results.

Milk is the most important single food used in gastric feeding. Three pints should be given every 24 hours. It should be citrated, and if necessary peptonized. One raw egg should be added to each pint of milk. The caloric value of 3 pints of milk and 3 eggs is approximately 1,200. The addition of 2 oz. of 20 per cent cream to each pint of milk will add a further 300 Calories. The egg-milk-cream mixture will supply ample protein (about 75 grams) and fat (about 100 grams), and will more than meet the body's requirement for calcium. If it is found that the milk is not being digested – as indicated by flatulence, diarrhœa, and the passage of excessive quantities of curd in the stools – it may be partly replaced by casein hydrolysate given as a 10 per cent solution. 1·5 grams per kilogram body weight will usually be found adequate to maintain nitrogenous equilibrium (see p. 389).

To bring the caloric value of the diet up to 2,500, 250 grams of carbohydrate should be given in the form of sugar, glucose, lactose, syrup or thin gruel. The simplest way is to give 5 pints of a 10 per cent solution of glucose in water. This amount of fluid, together with the 3 pints of milk, will ensure a fluid intake sufficient to overcome severe dehydration. For tropical countries, however, the amount may require to be increased to over 10 pints for the first 24 to 48 hours. Ransome, Gupta and Paterson (1944) state that the inherent dangers associated with large infusions by the intravenous route were not encountered in a big series of severely dehydrated and acutely ill patients in the tropics, who received from 10 to 15 pints of fluid daily.

If, however, it is considered that a total fluid intake of 6 pints is sufficient, approximately 30 grams of glucose should be added to each pint of milk, and the remaining 150 grams of sugar given in 3 pints of 10 per cent glucose solution. If hypochloræmia co-exists with dehydration, 2 pints of the glucose solution should be given in the form of 10 per cent glucose saline. This will supply 10 grams of sodium chloride which, together with the salt in the milk, should be sufficient to correct any existing deficiency. If gastric feeding has to continue for more than a few days, it is wise to implement the intake of vitamins C and B by adding daily one or two oz. of orange juice or 50 mg. of ascorbic acid, and a preparation of yeast, to the fluid in the reservoir of the drip apparatus.

2. Duodenal and Jejunal Feeding

If the tube is allowed to pass through the pylorus into the smal intestine, it is claimed by certain authorities that it is possible t maintain the nourishment of the patient and at the same tim secure complete gastric rest. Accordingly, duodenal and jejuna feeding have been advocated for the treatment of peptic ulcer and as a measure for the pre- and post-operative treatment o malnourished patients with gastric carcinoma.

It is doubtful whether the reasons advocated for these pro cedures are sound. For instance, it has been shown that when th stomach is empty it exhibits more motor activity than when i contains food. Furthermore, the remarkable control of hyper acidity which can be secured by continuous feeding through th stomach tube would not be achieved. Accordingly, we do nc recommend small-intestinal feeding for peptic ulcer. The sma intestine was never intended by nature to receive food directly int its lumen without previous chymification and digestion, and henc is apt to respond to abnormal stimuli by excessive peristalsis. A a result colic, flatulence and diarrhœa are easily produced, and th small-intestinal hurry which occurs leads to a failure in absorptio of nutrients.

Direction for Preparation

If jejunal feeding is undertaken, the strength of the suga solutions given should not exceed 10 per cent, while protein shou be given in the form of peptone, and milk should be previous peptonized. The Ivy-Wolfe formula which meets these requir ments is as follows: Water, 2,000 c.c.; cane sugar, 150 gram peptone powder, 80 grams; flour, 120 grams; milk, 1,500 c.c.; cream (20 per cent) 500 c.c.; salt, 16 grams. Total volume 4,000 c.c.

The sugar and peptone powder are dissolved in water and heated for several minutes. The flour is made into a smooth paste with the milk (previously peptonized) and brought to the boil. The two mixtures are combined and stirred vigorously. The pabulum is strained and stored on ice. Supplementary vitamins can be added.

Administration

About 50 c.c. of the pabulum is allowed to drip into the jejunum for 20 minutes followed by 40 minutes rest. This is repeated every hour. The amount of the pabulum is gradually increased according to the tolerance of the patient, until about 2,000 c.c. are entering the jejunum every 24 hours. In our opinion the method of intravenous alimentation with casein hydrolysates and glucose described on p. 388 is a more effective pre- and post-operative measure for the relief of malnutrition in patients suffering from serious gastric disease than is feeding through the jejunal tube, and hence should be the method of choice in the future.

3. Gastrostomy Feedings

If inflammation of the pharynx or gullet is so severe, or obstruction of the œsophagus so complete, that a soft rubber tube cannot be passed into the stomach, gastrostomy must be considered. If there is any reason to believe that the lesion is temporary, parenteral or rectal feeding should be instituted. If the lesion is likely to be present for weeks or is permanent, *e.g.* inoperable carcinoma of the œsophagus, operation should be proceeded with immediately, and a tube inserted into the stomach. The principles underlying the types of food for gastrostomy feeding are similar to those described under gastric feeding by the trans-nasal route. Milk, eggs, gruel, sugar and syrup are the basic articles of diet. Feeds should be given every two hours, and the amount administered at each feed should at first be approximately 4 oz., increasing gradually to 8 oz.

Composition of the Feeds

Since the patient does not taste his food, there is no particular point in flavouring his meals or using a large variety of foods. The diet must be adequate in Calories, fluid and all essential nutrients. Shortly after operation, when the stomach is contracted, it may not be possible for it to hold more than 4 oz. at a feed, but this amount should be gradually increased to at least 8 oz. as soon as possible.

When 4 oz. only can be given at a time, it is obvious that the feed will have to be spaced at hourly intervals if the patient's fluid an nutritional needs are to be met. When 8 oz. or more are retained the intervals between feeds may be increased to 2 or even 2½ hour between 7 a.m. and 10 p.m.

A pabulum of the following foods will meet the daily requir ments, and should be made up and kept in the refrigerator: mil 3 pints, or dried whole-milk powder, 8 oz., made up with wat to 3 pints; 3 eggs; 40 per cent cream, 2 oz.; sugar, 4 oz. or golde syrup, 5 oz. or wheat, corn or oatflour, 6 oz.; dried yeast, ½ oz salt, 1 teaspoonful; 2 oz. of fresh orange juice or 1 tablet of ascorb acid should be added to each of two feeds in the day. Liver jui or purée should be given on occasions, and if an iron deficien anæmia is present, 15 grains of iron and ammonium citrate shou be added to four or five of the feeds. The diet will contain approx mately 200 grams of carbohydrate; 80 grams of protein; and 1 grams of fat, giving a caloric value of 2,000 and a fluid conte of approximately 1,800 c.c. If additional Calories are require extra cream and sugar can be given. If this amount of fluid found to be greater than can be accommodated by the stoma in the 24 hours, the liquid milk ration should be reduced to pints, which should be reinforced with 2½ oz. of dried whole-mi powder.

4. The Nutrient Enema

Although Voit and Bauer published their observations on nutrie enemata as long ago as 1869, clinicians still differ widely as to t value of this special feeding method. The results obtained different workers in this field and by the same workers in differe patients are frequently contradictory. These divergent findi can be attributed partly to the different methods of investigati employed and the varying composition of the enemata used, a partly to the undoubted fact that in a proportion of individu the ileocæcal valve is incompetent, with the result that t nutrients reach the ileum from which absorption is known to oc far more freely than from the colon and rectum. This abnormal is too inconstant, however, to be relied on for therapeutics p poses. Fats, proteins, and carbohydrates in the form of cre milk, minced meat, sugar and cereals have all been recommen as constituents of the nutrient enema. It would appear desira accordingly, to discuss the evidence in favour of their employm

Fat and Protein

A study of the literature warrants the definite statement that fat and protein are absorbed from the rectum and colon too poorly to justify their use in the nutrient enema. If protein in the form of casein is submitted to prolonged digestion (18 hours) with trypsin, a considerable amount of the predigested protein may be absorbed. There is, however, no reason to believe that the quantities which can be absorbed from the lower bowel are capable of materially reducing the negative nitrogen balance of a seriously ill patient. Whether this can be secured by the rectal administration of casein hydrolysates has as yet to be determined.

Glucose

Although there appears to be a widespread belief among clinicians that glucose is excellently absorbed from the lower bowel, the extensive and carefully controlled investigations of Mackenzie (1943) show that this is far from being the case. Glucose enemata given to 35 children in amounts of 200 c.c. and in strengths of from 5 to 20 per cent failed to alter the level of sugar in the blood removed at 30-minute intervals. Mackenzie came to the conclusion, however, that glucose was absorbed, for a considerable part of it had disappeared when the fluid obtained by a rectal washout at the end of the experiment was analysed. He showed that the loss could not be accounted for by bacterial activity in the rectum and colon. Moreover, the protein-sparing effect of glucose was shown to have occurred, since excretion of nitrogen in the urine was diminished. It would appear therefore that glucose is absorbed by the lower bowel, but so slowly that the liver is able to remove it from the circulation before the blood sugar can rise. It is impossible to increase the absorption of glucose by raising the concentration of the solution. Concentrations over 10 per cent of glucose tend to give rise to rectal irritation followed by evacuation. It is doubtful if it is possible for the patient to obtain, under the best conditions, more than 500 Calories a day from rectal feeding. Accordingly, the idea that a patient's weight and strength can be maintained, far less increased, by nutrient enemata must be completely discarded. Rectal feeding has been described as 'subnutrition of a pronounced kind'.

Salt and Water

There is ample evidence that sodium chloride given in the form of normal saline is readily absorbed. Isotonic saline leads to no irritation of the lower bowel. Hypertonic saline, however, causes irritation and rapid evacuation of the fluid. The quantity of fluid which can be given per rectum varies greatly in different persons, depending on personal idiosyncrasy to rectal stimulation and on the method employed. In the most favourable circumstances 2,000 to 3,000 c.c. may be given in 24 hours.

Indications

Nutrient enemata are of value in the treatment of moderat degrees of dehydration and hypochloræmia. Rectal feeding i contra-indicated when diarrhœa or rectal disease is present o where the patient is unable or unwilling to co-operate.

Technique

If adequate quantities of fluid are to be given per rectum, clos attention to details of technique is required, especially the avoid ance of distension of the rectum. The special apparatus describe originally by Murphy is recommended.

Before beginning the administration of fluid, the lower bow must be emptied by a saline washout, and this should be done onc each day during the period of rectal feeding. The apparatu consists of a wide-bore rectal nozzle of glass or vulcanite which attached to a glass **Y**-tube, one arm of which is connected by thic walled rubber tubing to the drip-apparatus. The other arm connected with a piece of rubber tubing which acts as a free lim for the escape of flatus. The tubing near the nozzle is secured the thigh by a bandage, while the fluid container is attached a stand about 12 in. above the level of the rectum. The rate flow must be less than that which causes rectal distension a hence varies from case to case. It should be started at about drops a minute, and gradually increased to about 40. The high rate is equivalent to about 150 c.c. of fluid per hour. The fl may be stopped for one hour in four, the nozzle being left position. If the Murphy apparatus is not available, from 200 300 c.c. may be run very slowly into the rectum through a fun attached to a soft rubber catheter once every 3 or 4 hours. T patient should lie on his side with his buttocks raised on a pill

CONCLUSIONS

Special feeding methods should be considered as emergency p cedures for combating states of nutritional deficiency which can be corrected by the ingestion of food and fluid by the nor channels. They may be used to replace completely or to sup ment normal oral feeding. With the exception of gastric feeding through a tube, special feeding methods, because of inherent technical difficulties, can be used only for short periods, *i.e.* 3 days to 3 weeks. While all the methods described are of great value in restoring fluid and salt, feeding by the stomach tube is the only method which can supply all the nutrients required for the complete restoration and rehabilitation of a severely malnourished patient. Intravenous infusions of glucose will only supply the basal energy requirements, while the addition of adequate amounts of casein hydrolysates to the infusion will secure nitrogenous equilibrium. Subcutaneous and rectal infusions are only of value in counteracting moderate degrees of salt and fluid deficiency. The addition of glucose to the rectal infusion will supply less than one half of a patient's basal energy needs. It will thus be apparent that, while special feeding methods are undoubtedly valuable therapeutic procedures for rectifying critical states of nutritional deficiency in certain circumstances, their limitations must be appreciated. The correct selection of the method to be employed is dependent on a sound knowledge of these limitations and the ability to assess critically the type and degree of the nutritional deficiency present.

PART V

TABLES, RECIPES AND DIET SHEETS

CHAPTER XXI

FOOD TABLES IN THE ANALYSIS AN CONSTRUCTION OF INDIVIDUAL AN GROUP DIETS

IN Chapters II to VI, where the fundamental nutritional requir ments have been enumerated, and in Chapters XI to XIX, whe the principles of dietetic treatment in disease have been discusse various quantitative estimates have been given of the amounts the different nutrient factors which should be present in the di both in health and in disease. It follows therefore that a knowled of the chemical composition of foodstuffs is essential, not only the dietary treatment of disease, but also in assessing the adequa for health of the diet eaten by individuals or by groups.

Unfortunately, foodstuffs are not like chemical compounds wi a constant composition. Variations, sometimes considerable, a to be found in the composition of individual foodstuffs, especial of plant foods, depending on factors such as variety, state growth, and the climate and soil of the region in which the food w produced. Therefore, if a very accurate estimate of the intake any particular nutrient or nutrients is required, it follows that t only possible method is the chemical analysis of samples of t foodstuffs comprising the diet being studied.

While such chemical analysis of diets is essential in research the absorption and utilization of nutrients it is obviously too co plicated and tedious for the everyday practice of dietetics, where the nutritive value of diets must be calculated from tables givi the chemical composition of foodstuffs. From what has been sa about the variability in the composition of individual foodstuf it will readily be appreciated that food tables of this nature c give only average values or ranges of values for the amounts of t different nutrient factors present in any given foodstuff, and t reliability of the food table will therefore depend on the number samples of the different foodstuffs analysed to obtain the avera values.

Calculation of the nutritive value of diets by the use of food tables must therefore be regarded as giving only an approximation to their composition. With reliable food tables, however, this approximation can be remarkably close, as was demonstrated by Widdowson and McCance (1943) who, in a critical appraisal of the accuracy of their food tables, compared the figures for various nutrients in diets obtained by calculation from these tables with those obtained by chemical analysis of the diets.

TYPES AND USES OF FOOD TABLES

Tables giving the chemical composition of foodstuffs are necessary in several circumstances, *e.g.*:

(*a*) In prescribing diets for patients whose treatment requires quantitative control of the diet.
(*b*) In arranging diets for the group-feeding of persons in institutions, or rations for members of the fighting services.
(*c*) In assessing the results of dietary surveys undertaken to study the intake of essential nutrients by individuals or groups.

The type of food table required will not be the same for all the above purposes. Thus, when an individual diet is prescribed for a single person, foodstuffs which require cooking will be weighed or otherwise measured and served in the cooked form. In the process of cooking, the chemical composition of food is altered – for instance, changes in moisture content usually occur, and nutrients, especially vitamins, may be destroyed or extracted into the cooking water. Therefore, what is wanted for individual diets is a table showing the nutritive value of cooked foodstuffs.

On the other hand, when a diet is drawn up for an institution or a ration scale authorized for any of the fighting forces, the amounts of foodstuffs laid down represent the weights of the raw foodstuffs as purchased. Many foodstuffs in the form in which they are sold or issued contain inedible waste such as the bones in meat or the stalks of vegetables. Therefore, in calculating the nutritive value of a diet from the raw foodstuffs, allowance must be made for this inedible waste. In food tables, this is done in one of two ways:

(*a*) The nutritive value of the raw 'as purchased' weight of the foodstuffs is given, with allowance already made for any inedible waste.

(*b*) The nutritive value of the 'edible portion' of the foodstuff is given, with average figures for the percentage of inedibl waste in each of the individual foodstuffs.

In the latter case, the nutritive value of the 'as purchased' weigh of any foodstuff is easily calculated by first deducting the amoun of inedible waste from the 'as purchased' weight; this gives th amount of edible material in the 'as purchased' weight of foodstuf and this corrected weight can then be used in conjunction with table giving the nutritive value of the 'edible portion' of th foodstuffs.

The former method is of course the simplest, but there a complicating factors: for instance, meat may be sold or issue either boneless or with bone. Most tables of food values give th nutritive value of the edible portion only, and in using such tabl the weight of inedible waste in foodstuffs must be deducted fro the 'as purchased' weight, as described above.

In dietary surveys the weights of foodstuffs obtained will oft be the raw 'as purchased' weights, and for this type of work th procedure is the same as in drawing up group diets.

Although tables of the chemical composition of raw foodstuf are used in calculating the nutritive value of group diets or th results of dietary surveys, the food will of course be cooked befo reaching the persons comprising the group, and it is the nutrien contained in the cooked, not in the raw food which are actual consumed by these persons.

To assess the true nutritive value of a group diet, it is therefo necessary to make arbitrary deductions from the nutrients prese in the raw foods to allow for cooking losses and plate wastag Details of how this is done are given in the paragraph dealing wi group dietary analysis (see pp. 407 to 410).

To assist readers who may wish to prescribe diets differing composition from those included in this book, we have drawn Table 17 giving the chemical composition of the commoner Briti and foreign foodstuffs. This table, which is based chiefly on t Medical Research Council's War Memorandum No. 14, 'Nutriti Value of Wartime Foods' (H.M. Stationery Office, London, 194 will be found on pp. 427 to 435.

Explanatory notes for the use of Table 17 are given on pp. 4 to 439. Before using the table in drawing up diets or assessing t results of dietary surveys, the reader is strongly urged to read th notes carefully.

The table has been designed for use in calculating the nutri value of both individual and group diets, and therefore gives the chemical composition of one ounce of the edible portion of both cooked and raw foodstuffs and the average percentage of inedible waste. To avoid possible confusion, the nutritive values of foodstuffs as purchased are not included, but those can easily be calculated by the method described on p. 402.

Of the minerals, figures have been given only for calcium, iron, and sodium chloride. As explained in Chapter V, calcium and iron are the two minerals most likely to be deficient in a diet, while the sodium chloride figures have been inserted to enable the salt content of the diet to be calculated where required – as, for instance, in hot climates or where a salt-poor diet is being prescribed.

Figures have been given for the content of all the vitamins whose importance in human nutrition has been universally accepted and for which a reasonable estimate of the human requirement has been made. Recently isolated vitamins such as pantothenic acid and biotin have not been included, although figures for the amounts of these vitamins in foodstuffs are now appearing in the literature. The need for these vitamins in human nutrition still awaits final proof, and the quantitative requirements have not yet been adequately determined.

It has already been stressed that individual foodstuffs vary in chemical composition and that changes in the composition occur in cooking. These remarks apply with particular force to the vitamin content. For this reason it must be emphasized again that the figures given for the amounts of vitamins in foodstuffs should not be regarded as absolute, but as averages over a range of samples. As regards riboflavin and especially nicotinic acid, the analytical methods employed have not reached the same stage of reliability as in the case of the other vitamins, and the number of analyses so far published have been much fewer. The figures for nicotinic acid especially must therefore be regarded as tentative and provisional. Most of the published figures for riboflavin and nicotinic acid have come from America, and the amount of these vitamins in the corresponding foodstuffs produced in Great Britain may in many cases be somewhat different.

For the majority of diets likely to be used in medical practice, accurate prescription on a quantitative basis may only be required for one or at most a few of the essential nutrients (*e.g.* a low protein diet in acute nephritis). The need for including such a comprehensive food table in a book devoted primarily to medical dietetics may therefore be queried. But it should be pointed out that the nutritional requirements of a patient are as great as, and in some respects may be greater than, those of a healthy person, and it is unsound practice to prescribe a diet which conforms to the requirements for the condition under treatment but is deficient according to the normal standard in one or more of the other essential nutrients. Adequate nutrition both in health and disease requires that all the essential nutrients should be present in the amounts required for health, and therefore every diet which has to be taken for more than a few days on end should have its content of all the essential nutrients checked against known phsyiologica requirements.

ANALYSIS OF INDIVIDUAL AND GROUP DIETS

Information about the composition of the diet may be requirec with respect either to an individual diet or to the average die consumed by a group of persons receiving their food from the sam source.

In medical dietetics, where diets are prescribed and served fo individual patients, we have an example of dietary analysis by th individual method, although, as described below, a somewha similar procedure is the most accurate method of assessing a individual's food intake short of chemical analysis.

Since the group method of dietary analysis is used in assessin the adequacy of institutional diets, for instance the ordinary die issued in hospitals to patients not on special diets, it is not out c place to discuss this method in a book dealing chiefly with die in disease.

Individual Dietary Analysis

To estimate the composition of a diet, the most accurate metho is of course to record the weight of every food actually eaten ov a period, and to use these weights in calculating the nutritive valu of the diet from a table which gives the composition of foodstuf as eaten.

The first thing to be settled in carrying out such a survey is tl length of time over which it will be undertaken. The longer tl period the more accurate will be the result, as the effect of day-t day fluctuations in the diet will be minimized: and it is safe to sa that, to obtain anything like an accurate picture of the individual diet, the period of the survey should be at least one week.

Every item of food to be eaten must be weighed on an accurate balance weighing at least to $\frac{1}{8}$ oz. Foods should be weighed on a light plate and transferred from there to the ordinary plate. Any food left at the end of the meal must similarly be weighed, the waste from each individual foodstuff being weighed separately. The amount of each food actually eaten will of course be represented by its weight as served less the weight of any left on the plate at the end of the meal.

If it can safely be assumed that only the subject of the survey will partake of the food, items such as milk, jam, butter, and sugar may be weighed in their domestic receptacles at the beginning and end of a meal or day.

Apart from the weights, the record must of course include an accurate description of the foodstuffs themselves; this is very important in the case of cooked dishes where the type of food and the method of cooking must be mentioned. It is equally important to record whether the food used in preparing each dish was fresh or processed.

It will be obvious, when the following paragraphs on group dietary analysis have been read, that individual dietary analysis carried out on the lines described above is much the more accurate method, since the results can be interpreted without the difficulties arising in group dietary analysis from the use of arbitrary assumptions and factors which tend to interfere with the significance of the results.

The serving of special diets to patients according to a prescription laying down the type and amounts of food to be consumed is, as it were, the process of individual dietary analysis in reverse.

Group Dietary Analysis

When a group of persons is fed from one central source of supply, as in a family or an institution, an estimate of the average diet consumed by individual members of the group can be made from a knowledge of the amount of food entering the family larder or institution food store over a period and the amount of waste over the same period. A less accurate estimate can be arrived at when the amount of waste cannot be weighed. Both methods are described below.

1. *Group Dietary Analysis with Measurements of Food Wastage*
   This method involves the following procedures:
   (*a*) The amount of food supplied to the group must be ascertained from the following records:

(i) The amount of each foodstuff in store at the beginning of the survey period.

(ii) The amount of foodstuffs entering the food store during the survey period.

(iii) The amount of foodstuffs remaining in store at the end of the survey period.

The amount of food supplied to the group during the survey period will be obtained for each foodstuff by adding the amounts in store at the beginning of the period to the amounts entering the store during the period and subtracting the amounts in store at the end of the period.

(*b*) The amount of food waste must be measured and deducted from the gross amount of food supplied.

Where food waste has been collected and weighed in different categories – *e.g.* meat bones, bread, fats, potato peelings, vegetable leaves and stalks – the amount of waste in each category should be deducted from the weight of the corresponding foodstuff supplied over the period which has been ascertained by the method described in sub-paragraph (*a*) above. The corrected weight of foodstuffs therefore represents the edible portions of the raw foodstuffs which have been consumed by the group over the period of the survey.

Where the food waste is not separated into categorie and only the total weight of waste is known, the weight o the edible portion of the food supplied will have to b estimated by the less accurate method of expressing th ascertained weight of food waste as a fraction of the tota weight of all foods supplied and deducting this fractio from the weight of each individual foodstuff consumed.

(*c*) The next step is to divide the weight of the edible portion o each foodstuff consumed by the number of days in th survey period and by the number of persons in the grou so as to obtain the average daily food consumption pe person. This procedure as it stands will only give a reasonable result if all members of the group are of the same ag and sex. But if the group consists of persons of differen ages and sexes, the complicating factor of varying foo intakes and requirements arises.

This complication has to be resolved by assuming th women and children consume a definite fraction of what i eaten by the average adult male. To each woman and t children of different sex and age a certain fraction known as the 'man value' is assigned, the 'man value' being in fact the arbitrary estimate of the fraction of an average adult male's consumption (taken as unity) which is likely to be eaten. If the sum of the 'man values' of a group is used in dividing the total food intake, the resulting figure and also the nutritive value of the diet calculated from this will be expressed in terms of the intake of an average adult male. Therefore by setting these figures against the dietary standards of an adult male, an approximate estimate can be obtained of the adequacy of the diet provided for a group of persons of different sexes and ages.

This system of 'man values' relates only to the total amount of food eaten, that is to the Calorie value, and therefore does not make allowance for the special needs of certain groups, *e.g.* the increased calcium requirements of children or pregnant or lactating women, or the relatively increased protein requirements of children as compared with those of an adult male.

Unfortunately the estimation of the 'man value' being an arbitrary procedure, different authorities have laid down different scales of 'man values'. Among the most recent of the scales is that given by the Technical Commission of the Health Committee of the League of Nations (*The Problem of Nutrition*, Vol. II, Report on the Physiological Basis of Nutrition, 1935) and set out in Table 13.

TABLE 13

SCALE OF 'MAN VALUES'

	Co-efficient ('man value')
Child aged 1–2 years	0·3
,, 2–3 ,,	0·4
,, 3–4 ,,	0·5
,, 4–5 ,,	0·5
,, 5–6 ,,	0·6
,, 6–7 ,,	0·6
,, 7–8 ,,	0·7
,, 8–9 ,,	0·7
,, 9–10 ,,	0·8
,, 10–11 ,,	0·8
,, 11–12 ,,	0·9
,, 12 and over	1·0
Man or Woman	1·0

This scale makes no allowance for the increased requirements of adolescents resulting from rapid growth and physical activity, or for the lesser requirements of an adult woman as compared with an adult man. In our opinion the above scale of 'man values' might well be modified to allow for these two cases. We suggest that for boys of 13 to 15 years of age the co-efficient should be 1·1; for adolescent males of 16 to 20 years 1·2 to 1·3; and for adult women 0·8 to 0·9.

Therefore in assessing the diet provided for groups of persons of different sexes and ages, a 'man value' must be ascribed to each individual, and the total of these 'man values' should be used when reducing the total food intake to the intake per person.

(*d*) Having calculated the daily food intake of individual members of the group (either directly where they are all of the same sex and age or indirectly by the application of 'man values), the final step is to calculate the nutritive value of the diet from a table of food values giving the composition of the edible portion of raw foodstuffs. The result is of course the potential nutritive value of the raw foodstuffs of the diet. But the diet has been eaten in cooked form, and before we can assess the true nutritive value obtained from the diet by each member of the group, some arbitrary estimate must be made of the losses of nutrients occurring in cooking. The vitamins are the only class of nutrients in which such an adjustment is necessary – the following deductions (see Table 14) from the vitamin content calculated from the figures for raw foodstuffs representing, in our view, the approximate cooking losses in an average mixed diet:

TABLE 14

Percentage Loss of Vitamins in Cooking

	Deduction for cooking loss per cent
Vitamin A	5
Vitamin B_1	15
Riboflavin	10
Nicotinic Acid	up to 20
Ascorbic Acid	60

When the vitamin intake has been corrected for losses in cooking, the adequacy of the diet of the individual can be estimated by comparing its composition with the dietary standards laid down in Table 1 (p. 2) for the group being studied. If 'man values' have been used in estimating the composition of the average diet, the standard of comparison will of course be the nutritional requirements of the adult male.

2. *Group Dietary Analysis without Measurement of Food Wastage*

This is a less accurate method than the one described above, but dietary analysis by the group method being at best only an approximation, we consider that it is justifiable to apply the method even where the amount of food waste cannot be ascertained.

The steps involved in this simplified method are as follows:

(*a*) The amount of food supplied to the group whose diet is being studied is calculated in exactly the same way as in the method described in paragraph 1 (*a*) above.

(*b*) Allowance is now made for waste, first for inedible matter and then for cooking and table wastage:

(i) *Inedible matter.* Where applicable, a deduction for inedible waste must be made from the total weight of each foodstuff supplied, the amount to be deducted being ascertained by reference to the figures for the average percentage of inedible waste given in the table of food values. The result gives the weight of edible raw foodstuffs supplied. Where the food table being used gives the composition of the raw foodstuffs as purchased, allowance has of course already been made for inedible waste, and the above step in the calculation is accordingly omitted.

(ii) *Cooking and table wastage.* It is impossible to give even an arbitrary figure for such wastage, which will vary with the cooking methods employed, the feeding habits, and the dining-hall discipline among the group being studied. Figures up to and even over 10 per cent have been recorded for table wastage in institutions, and the reader must judge from his own observations of the extent of table wastage, etc., whether he should employ this figure or a lesser one. The figure of 10 per cent is, however, the conventional one for such waste. Whatever percentage figure is finally decided upon, a corresponding deduction must be made from the weight of the edible raw foodstuffs supplied.

The resulting figures are the weights of the foodstuffs actually consumed by the group, and the subsequent steps in the analysis will therefore be exactly the same as those described in paragraphs (*c*) and (*d*) above for group dietary analysis where food wastage is measured.

THE CONSTRUCTION OF DIETS

Much of this chapter has been devoted to the use of food tables in dietary surveys. Of more importance in practical dietetics is a knowledge of how to draw up a diet for an individual patient or for groups of persons in institutions, etc.

There are five main steps in the construction of a diet scale:

1. Decide how many Calories the diet must contain. Unless for special reasons, such as the restriction of Calories in an obesity diet, the number of Calories prescribed should correspond with those shown in tables of dietary standards as appropriate for the age, sex and physical activity of the individual concerned. (See Table 1 on p. 2.)

2. Decide what proportion of the Calorie intake should be from protein, fat and carbohydrate respectively. The usual allotment is:

10 to 15 per cent of Calories from protein
30 to 35 per cent of Calories from fat
55 per cent of Calories from carbohydrate.

These proportions should be adhered to approximately unless there are special dietetic reasons to the contrary, such as the restriction of protein in a nephritic diet, of fat in a diet for jaundice, or of carbohydrate in a diabetic diet. In such cases the weight in grams of protein, fat or carbohydrate to be allowed to the patient must be decided, and the Calories obtainable from this weight of the nutrient must be subtracted from the total number. The remaining Calories must then be distributed over the other two nutrients. But unless there are special indications, the proportion of 10 to 15 per cent of the total Calories from protein should be maintained.

3. Ascertain the number of grams of each of the three classes of foodstuff which the diet must contain. This is done by dividing the number of Calories to be derived from protein, fat and carbohydrate by 4, 9 and 4 respectively.

4. Translate this theoretical diet of grams of protein, fat and carbohydrate into a practical diet of so many ounces of different foods per day.

Consideration must be given at this stage to certain factors such as the approximate amount of each foodstuff which a person might be expected to eat daily and the types of foodstuffs from which a suitable daily menu can be provided. This will involve the provision of sufficient alternatives to certain of the items to allow for variety from day to day.

An important consideration when dealing with individual diets is that the patient's dietary prejudices must be respected wherever this does not interfere with his treatment. Prescription of items or amounts of food which the patient will reject defeats the object of dietary treatment, in that the patient will not be receiving the nutrients considered necessary for his condition. Therefore before drawing up an individual diet, the doctor or dietitian should always find out from the patient what his special likes and dislikes are, and should endeavour to allow for these in constructing the diet, provided always that they do not offend against the canons of sound nutrition.

It is essential at this stage to know the approximate amounts of the different categories of foodstuff to prescribe either as servings at individual meals or as the daily ration. These are enumerated in Table 15 (p. 412), which gives the average amounts for an adult male; women and young children will require less. Where applicable, the amounts refer to cooked food.

If the diet being arranged is an individual one, made-up dishes such as puddings and pies can of course be included: 3 to 4 ozs. is a generous helping of such dishes.

With these individual servings or daily portions as a guide, the diet can now be constructed in terms of actual foodstuffs, the amounts of protein, fat, carbohydrate, and Calories in each of the foodstuffs added to the diet being set down in tabular form. It is best to begin with protein and to start with the animal foodstuffs, prescribing for an ordinary diet sufficient servings of these to supply 40 to 50 grams of first-class protein. The rest of the protein can be left for the moment – it will be made up from the cereals and vegetables. Fatty foods may now be added to bring the total fat content of the diet up to the amount decided upon; and lastly, the carbohydrate foods should be added in sufficient amount to provide the required carbohydrate content. These carbohydrate foods will also supply the remainder of the protein, and slight

TABLE 15

Servings at Individual Meals or Per Day

Item	Portion
(*a*) *Protein Foods*	
(i) Meats	2 oz. – small serving 3 oz. – average serving 4 oz. – large serving
(ii) Fish	3 oz. – average servin 4 oz. – large serving
(iii) Offals	4 oz.
(iv) Sausages	4 oz.
(v) Cheese	¾ to 1 oz. (daily)
(vi) Milk	½ to 1 pint (daily)
(*b*) *Fatty Foods*	
(i) Bacon	2 oz.
(ii) Butter or margarine	1½ to 2 oz. (daily)
(*c*) *Carbohydrate Foods*	
(i) Bread	8 to 12 oz. (daily)
(ii) Cakes or buns	2 to 3 oz. (daily)
(iii) Flour for cooking purposes	1 to 2 oz. (daily)
(iv) Cereals (rice, oatmeal, etc)	1 to 2 oz. (daily)
(v) Sugar	2 oz. (daily)
(vi) Jam	1 to 2 oz. (daily)
(vii) Fresh fruit	4 to 6 oz. (daily)
(viii) Fresh vegetables	4 oz. – average servin 6 oz. – large serving
(ix) Potatoes	4 to 6 oz.
(x) Dried pulses (cooked)	3 to 6 oz.
,, ,, (raw)	1 to 2 oz.

adjustments may if necessary be made in the amounts of anima foodstuffs prescribed in order to bring the total protein content t the desired level.

The list of foodstuffs so obtained must now be arranged in th form of a daily menu. In most individual diets for patients, it i unnecessary to lay down a definite scale of ounces of each foodstu per serving or per day. Except where a rigidly quantitative diet i essential, the number of ounces of foodstuffs allotted to the die when calculating its nutritive value as described above, may b translated into servings or common measures along the lin indicated in Table 24 on p. 445.

If, on the other hand, the diet is being drawn up for a group persons in an institution, the amounts of each foodstuff will hav to be given as a definite scale of so many ounces per head per da so that the caterer may ascertain the amount of food he must ord by multiplying the scale per head per day by the number of individuals to be fed. Since the food purchased by the caterer will be in the raw state, allowance must be made for inedible waste when drawing up the diet scale. For example, if it has been decided that each person should receive 6 oz. of potatoes daily, reference to the column headed 'Per cent inedible waste' in the table of food values on pp. 427 to 435 will show that the amount of raw potatoes required to provide this amount is about 8 oz., and it is the latter figure which must be inserted in the diet scale supplied to the caterer.

5. The final step is to calculate the amount of minerals (calcium and iron) and vitamins in the diet, and to adjust the amount of protective foods prescribed in order to ensure an adequate mineral and vitamin content. For convenience, the richest food sources of the important minerals and vitamins are summarized here:

Calcium. Milk and cheese are by far the most important sources. Tinned fish provides a fair amount of calcium because of the bones which are eaten with it. Vegetables are often mentioned as a source of calcium, but reference to the amounts considered to be a reasonable serving and to the table of food values will show that the importance of vegetables in this respect has been exaggerated.

Iron. Liver, wholemeal cereals, pulses, and meat.

Vitamin A. Liver, butter, vitaminized margarine, cheese, milk, green leafy and yellow vegetables.

Vitamins of the B group. Wholemeal cereals, nuts, pulses, green vegetables, liver, ham, other meats, and milk and cheese (riboflavin only). Yeast is the richest source of the B vitamins.

Vitamin C. Certain fresh vegetables and fruits.

It is important to remember that the concentration of minerals and vitamins per unit weight of a foodstuff is not the only factor determining its importance in the diet. The amount of the foodstuff normally eaten must also be taken into account. For example, parsley is a very rich source of both vitamin A and vitamin C, but the amount of this vegetable ordinarily consumed can make only an insignificant contribution to the vitamins A and C content of the diet. Conversely, among the vegetables, potatoes have a low vitamin C content, but since they are normally eaten daily, and in relatively large amounts, they supply a large proportion of the vitamin C intake.

After studying the principles of dietetic treatment given in Part IV, it should be possible for the doctor to construct his own diet sheets with the help of the tables given in the following pages.

Experience has shown, however, that, through lack of culinary knowledge, familiarity with food tables or time, most practitioners are unwilling or unable to undertake this task. It is for this reason that ready-made diet sheets are supplied which will cover all dietetic requirements under ordinary circumstances.

Food Exchanges

Since monotony is a potent factor in causing dietetic instructions to be broken, it is essential that the patient, especially if he is diabetic, should be supplied with an adequate number of food exchanges. The principle on which such exchanges have been arranged is as follows:

Protein Exchanges

Breakfast: The basis of the breakfast exchange is one egg or one egg and 1¼ oz. of bacon.

Mid-day Meal: The basis of the lunch exchange is 3 oz. of meat.

Evening Meal: The basis of the dinner and high tea exchange is 3 oz. of white fish.

A study of Table 25 (p. 446) will indicate the wide variety o exchanges available. It will be noticed that butter or vitaminized margarine must be added to or subtracted from the daily ration o these products if the caloric value as well as the protein content o the various exchanges are to be identical. It should be pointed out, however, that it is not necessary to measure accurately such alterations in the ration. Once the patient has been taught to recognize what a ¼ oz. pat of butter or margarine looks like, the alterations can be made by sight.

Carbohydrate Exchanges

Of particular importance in the treatment of diabetes are the carbohydrate exchanges. The basis of these is 1 oz. of bread (15 grams carbohydrate). Throughout the diet sheets bread is prescribed in 1 oz. quantities or multiples thereof. It is extremely simple, therefore, for the diabetic patient to replace 1 oz. of bread in his diet by ¾ oz. of oatmeal or 3 cream crackers or 5 oz. o orange or 2½ oz. of potato whenever he wishes, as each of these helpings contain 15 grams carbohydrate. Similarly, when porridge is prescribed, the amount of oatmeal recommended is always ¾ o or multiples thereof so that, if the patient does not like porridge he can replace it with an ounce of bread or any of the carbohydrate exchanges mentioned above and given in detail in Table 25.

The vegetables and fruits are arranged in Tables 18 to 20 according to their carbohydrate content and in such a way that a simple calculation will permit the patient to have a wide variety of these articles in his diet.

Fat Exchanges

The basis of the fat exchanges is 1 oz. of butter. 1 oz. of butter or vitaminized margarine = 1½ oz. of bacon = 2 oz. of 40 per cent cream = 4 oz. of 20 per cent cream.

It is the duty of the physician to explain the bases of food exchanges to his patients and supply them with suitable tables.

100 Calorie Portions

If it is found that a patient on a 'maintenance' diet is either losing weight and strength or, alternatively, putting on too much weight, the caloric value of the diet will have to be increased or reduced respectively. At the same time it may be desirable or essential that the intake of one or other of the food constituents should remain unaltered. For instance, a diabetic patient who is satisfactorily stabilized in so far as his glycosuria is concerned, may be losing weight, in which case the doctor will wish to increase the caloric value of the diet without altering the carbohydrate portion. This can be done without any calculations by prescribing one or more additional 100 Calorie portions of fat or protein, as given in Table 21. Similarly, a nephritic patient on a standard protein intake, who was not maintaining his strength, could have the caloric value of the diet raised by the addition of one or more 100 Calorie portions of carbohydrate or fat. It is hoped that the Table of 100 Calorie portions containing standard amounts of carbohydrate, protein or fat will be of considerable help to the family doctor in making any necessary adjustments in his patients' diets.

Low Protein Dishes

Whenever the daily protein allowance is reduced to 60 grams or less, as for example in renal or cardiovascular disease of various types, technical difficulties arise in the construction of the diet. It is usually advisable, under such circumstances, to prescribe a vegetarian breakfast (cereal and fruit), allow a small helping of meat, chicken or fish at one meal, and an egg or equivalent at the third. The egg may be boiled, poached, scrambled or made into an omelette to which tomatoes, mushrooms or asparagu tips may be added. Since the equivalent of an egg in terms protein is approximately 1 oz. of meat, fish, or cheese, considerabl ingenuity is required to make this small quantity into an acceptable dish. The meat or fish can be cooked with potatoes, ric macaroni, etc., and served as a pie, rissole, soufflé or kedgere Cauliflower au gratin, macaroni and cheese, cheese custard, an Welsh rarebit suggest themselves as suitable dishes for utilizin the cheese ration. The low protein recipes given on pp. 419 to 42 will serve as examples. They are not suitable for diabetic diet because of the variable quantity of carbohydrate contained in th different dishes.

12-gram Carbohydrate Puddings

A pudding containing 12 grams of carbohydrate is a useful additio to the menus of diabetics. It can replace ¾ oz. of bread, 2 oz. potatoes or 4 oz. of any 10 per cent fruit. Suitable recipes are give on pp. 423 to 424.

High Carbohydrate, Low Fat Puddings

A pudding rich in carbohydrate and low in fat is of particul value when steatorrhœa is present, as occurs in diseases of the liv and biliary tract accompanied by jaundice, in diseases of th pancreas and in tropical and non-tropical sprue. They are als useful in all infective and toxæmic states. Suitable recipes will b found on pp. 422 to 423.

Soups

From the dietetic point of view, soups may be divided into tho of negligible food value and those of high food value. In the fir category are the clear soups and in the second the cream soups.

Clear Soups (consommé or bouillon) are broths made from beef chicken stock from which all fat has been carefully removed. Sma amounts of vegetables of very low caloric value may be boiled the stock, *e.g.* asparagus, cabbage, cauliflower, celery, etc. Becau of their negligible food content, clear soups are of value in th dietetic treatment of obesity and diabetes. Because of their co tained extractives, they are useful as stimulators of appetite a gastric secretion in asthenic individuals with hypochlorhydria achlorhydria. They are obviously contra-indicated in peptic ulce acid gastritis or hyperchlorhydric dyspepsia.

With a little ingenuity a variety of soups containing 5 gra of carbohydrate to the serving can be made by the addition to th stock of 3½ oz. of milk or an appropriate quantity of vario vegetables. It may be simpler for the patient to obtain such soups from a manufacturing firm. Thus Crosse and Blackwell market excellent soups in tins. Some varieties contain no carbohydrate, while others contain 5 grams per serving. Full instructions are supplied by the manufacturers in regard to the preparation of the soups for the table. If a diabetic patient selects a soup containing 5 grams of carbohydrate, he must deduct from his diet an equivalent amount of carbohydrate, *i.e.* $\frac{1}{3}$ oz. of bread, or 1 biscuit, or $1\frac{3}{4}$ oz. of 10 per cent fruit.

Cream Soups are made from meat or chicken stock, milk or a mixture of milk and stock. A rich purée of vegetable or cereal (artichoke, carrot, potato, cauliflower, barley, etc.) is cooked with the stock which is further enriched with cornflour, butter or margarine, milk or cream. The final product is therefore of high caloric value and devoid of mechanically irritating residue. Hence cream soups are of value in the treatment of peptic ulcer and hyperchlorhydria, and for the correction and prevention of debility and malnutrition which accompany and follow infective and toxæmic states. Because of their content of carbohydrate and fat, they are obviously contra-indicated in diabetes, obesity or steatorrhœa. Soups rich in carbohydrate and low in fat are of value in the treatment of hepatitis and obstructive jaundice. Soups rich in carbohydrate and low in protein are useful in chronic renal disease associated with a failure in the excretion of nitrogenous waste products but without œdema. The addition of a rich purée of potato, carrot, artichoke, etc., to a vegetable stock, which has been thickened with cornflour, will form a high carbohydrate soup. Eggs, milk and cream must not be used.

Suitable recipes for clear and cream soups will be found on pp. 424 and 425.

Weights and Measures

In the Tables, Recipes, etc., the following approximate equivalents are assumed:

30 grams = 1 ounce.
30 cubic centimetres = 1 fluid ounce.
1 kilogram = 2·2 pounds.
600 cubic centimetres = 1 pint.
100 grams = $3\frac{1}{2}$ ounces.
100 cubic centimetres = $3\frac{1}{2}$ fluid ounces.
1 litre (1,000 cubic centimetres) = 35 fluid ounces ($1\frac{3}{5}$ pints approx.).

Diets—Inexpensive and Expensive

It will be noticed that many of the diet sheets are given in duplicate, one being designed for persons of the hospital class and the other for persons in better financial circumstances. The two diets differ not only in their costs but also in the way in which the meals are arranged. The majority of working men have their main meal ('dinner') in the middle of the day, and on their return from work have a 'high' tea between 5 and 6 p.m., at which an egg, fish or meat dish is eaten. Most persons belonging to the business and professional classes prefer a small mid-day meal (lunch) and a light afternoon tea, and take their main meal ('dinner') at 7.30 to 8 p.m. It is essential, when prescribing a diet, that due consideration be given to the financial and social status of the patient.

RECIPES

Barley Water—Fruit Juice Recipe for Acute Nephritis (1st stage)

	Prot.	Fat	CHO	Calories
¾ oz. pearl barley 1 pint water } strained	1·6	0·4	15·5	72
¾ pint grape fruit juice	2	—	14	64
Juice 1 orange	0·7	—	8·4	36
Lactose, 5 oz.	—	—	135	540
Garnish with thin slice of lemon or sprig of mint.				
	4·3	0·4	172·9	712

Feeds to be given 2-hourly.

STONE GINGER BEER

Ingredients
1½ lb. white sugar.
½ oz. cream of tartar.
3 level teaspoons ground ginger.
2 quarts boiling water.
3 quarts cold water.
½ oz. brewer's yeast.

Method Into a stone jar put sugar, cream of tartar and ground ginger.
Add boiling water, and when all is dissolved, add cold water.
When just lukewarm add yeast mixed with a little of the liquid, and leave to work from 6 to 12 hours.
Skim and bottle.

This makes 5 quarts and costs 6d. (pre-war).

Note. One pint of this drink forms a pleasant way of adding 60 grams carbohydrate to any high-calorie diet.

LOW PROTEIN DISHES CONTAINING APPROXIMATELY 7·5 GMS. PROTEIN

Mutton and Macaroni Pie (for 2 people)

Prot. 15 Fat 20 CHO 19 = Calories 316

Ingredients
1⅔ oz. mutton.
2 oz. cooked macaroni.
4 oz. gravy.
½ oz. breadcrumbs.
½ teaspoon minced capers.
½ oz. butter (or vitaminized margarine).

Method Butter a pie-dish.
Arrange alternate layers of mutton, macaroni, gravy and capers.
Sprinkle with breadcrumbs.
Arrange small pats of butter or margarine on top.
Bake in moderate oven ½ hour.

Stuffed Tomatoes

Prot. 7·5 Fat 3·5 CHO 6·5 = Calories 87

Ingredients
1 large tomato – 4 oz.
¼ oz. breadcrumbs.
1¼ oz. minced lean boiled ham.
Pinch mixed herbs.
½ oz. finely chopped onion.
Salt and pepper.

Method
Slice top off tomato, and carefully scoop out centre.
Fill with ingredients well mixed together.
Place in greased tin and bake in moderate oven ¼ hour.

Fish Kedgeree (for 2 people)

Prot. 15 Fat 10 CHO 15 = Calories 210

Ingredients
2 oz. cooked fish.
2 oz. cooked rice.
½ hard boiled egg.
¼ oz. butter.
Salt and pepper.

Method
Melt butter and add rice and fish.
Stir well with fork and season with white pepper and salt.
Pile roughly on hot dish.
Arrange pieces of egg white round, and rub yolk through sieve on top.

Fish Pie

Prot. 7·5 Fat 9 CHO 7 = Calories 139

Ingredients
1 oz. cooked fish.
¼ oz. grated cheese.
2 oz. tomato.
¼ oz. butter or vitaminized margarine.
¼ oz. flour.
3 oz. fish stock.
Seasoning.

Method
Skin tomato and cut in slices and place in buttered pie dish.
Melt butter or margarine, add flour and cook slightly.
Add stock gradually, stirring all the time. Cook well.
Add flaked fish.
Add to tomato, and cover with grated cheese.
Place a small pat of butter on top and bake for ¼ hour.

Fish Soufflé (for 2 people)

Prot. 15 Fat 12 CHO 6 = Calories 192

Ingredients
¼ oz. flour.
¼ oz. butter or vitaminized margarine.
1½ oz. flaked fish.
1½ oz. fish stock.
1 egg yolk.
1 egg white.
Finely chopped parsley.

Method
Melt butter or margarine and add flour.
Add stock gradually and cook well.
Add seasoning, flaked fish and egg yolk.
Lightly fold in beaten white.
Steam in greased basin ½ hour.
Serve at once, garnished with parsley.

Fish in Baked Potato

Prot. 7·5 Fat 7 CHO 14·5 = Calories 151

Ingredients
1 baked potato – 3 oz.
⅛ oz. butter or vitaminized margarine
½ oz. cream.
Sole – 1 oz.
Seasoning.

Method
Scoop out potato.
Mix with butter or margarine or cream.
Place fillet of sole, which has been rolled and cooked in butter in oven, inside the potato, and surround with creamed potato.
Lightly brown in oven.

Cauliflower au Gratin (for 2 people)

Prot. 15 Fat 21 CHO 11 = Calories 293

Ingredients
Medium cauliflower.
¼ oz. butter or vitaminized margarine.
¼ oz. flour.
1⅓ oz. grated cheddar cheese.
3 oz. milk.
Salt, pepper, mustard.

Method
Cook cauliflower without breaking and stand in hot vegetable dish.
Mix roux of butter and flour.
Add milk gradually and cook well.
Season. Add egg and 1 oz. grated cheese.
Pour over cauliflower.
Sprinkle remainder of cheese on top.
Brown under grill.

Macaroni and Cheese

Prot. 7·5 Fat 15 CHO 14 = Calories 222

Ingredients
2 oz. boiled macaroni.
⅓ oz. grated cheese.
⅛ oz. breadcrumbs.
¼ oz. butter or vitaminized margarine.
Salt and pepper.
1 egg yolk.

Method
Add butter or margarine, cheese, seasoning and egg yolk to boiled macaroni.
Place in buttered pie-dish, sprinkle with breadcrumbs and brown in oven 20 minutes.

Cheese Pudding

Prot. 7·5 Fat 14 CHO 6·5 = Calories 180

Ingredients
¼ oz. breadcrumbs.
2 oz. milk.
⅓ oz. cheese.
½ egg.
¼ oz. butter or vitaminized margarine.
Salt and pepper.

Method
Mix breadcrumbs, grated cheese and seasoning.
Boil milk and butter or margarine, add to mixture and leave for ½ hour.
Add beaten egg.
Pour into buttered pie-dish and bake in slow oven for ½ hour.

HIGH CARBOHYDRATE LOW FAT PUDDINGS

APPLE MERINGUE

Prot. 1·5   Fat —   CHO 30   = Calories 127

INGREDIENTS
3 oz. cooked apple.
½ egg white.
1 oz. sugar.

METHOD
Sieve apple.
Lightly fold in egg white and sugar.
Pipe on to baking sheet and bake in very slow oven until set.

BAKED SEMOLINA PUDDING

Prot. 8   Fat 0·2   CHO 32   = Calories 162

INGREDIENTS
½ oz. semolina.
5 oz. skim milk.
¼ oz. brown sugar.
½ egg white.
Vanilla essence.
½ oz. strawberry jam.

METHOD
Sprinkle semolina into boiling milk and simmer for 5 minutes, stirring well.
Remove from heat. Add sugar and vanilla essence.
Pour into buttered pie-dish and bake in moderate oven till firm.
Cover with 1 dessertspoonful strawberry jam.
Pile stiffly beaten white of egg + 1 teaspoonful sugar on top.
Brown in oven.

BANANA AND RASPBERRY WHIP

Prot. 2   Fat 2   CHO 33   = Calories 158

INGREDIENTS
3½ oz. banana.
2 oz. raspberries.
1 digestive biscuit.
2 oz. lemon juice.
½ oz. glucose.

METHOD
Peel banana and mix with crushed raspberries.
Add lemon juice and glucose.
Place crushed digestive biscuit in bottom of glass.
Pile fruit pulp on top.

PEAR MELBA

Prot. 0·4   Fat —   CHO 24   = Calories 98

INGREDIENTS
2 halves tinned pear.
2 tablespoonfuls pear syrup.
2 tablespoonfuls strained strawberry jam

METHOD
Mix the pear syrup with the strained jam.
Put 2 halves of pear together in a glass dish.
Pour syrup over.

LEMON SAGO

Prot. 0·5   Fat 0·1   CHO 79   = Calories 319

INGREDIENTS
1 oz. sago.
½ pint water.
Juice of 1 lemon.
½ oz. sugar.
2 oz. syrup.

METHOD Boil sago until transparent.
Add other ingredients.
Pour into pudding dish.

12-GRAM CARBOHYDRATE PUDDINGS FOR DIABETES

All butter, cream and milk used in these recipes must be taken from the daily ration allowed in the diet sheet.

MILK PUDDING

INGREDIENTS ½ oz. cereal.
5 oz. milk from ration.
Add water if necessary.

The addition of 1 egg yolk and egg white beaten and folded into pudding gives a change.

APPLE SAGO

INGREDIENTS ⅓ oz. sago.
3 oz. stewed apple.
¼ teaspoonful grated lemon rind.
1 oz. lemon juice.
4 oz. water.
Saccharin.

METHOD Cook sago in water till clear.
Add stewed and sieved apple, sweetening and lemon rind and juice.
Pour into a buttered mould.

APPLE CUSTARD

INGREDIENTS 2 oz. apple.
4 oz. milk.
¼ oz. butter or vitaminized margarine.
½ egg.
1 teaspoonful glucose.

METHOD Stew apple in water and place in centre of buttered pie-dish.
Beat egg with milk and add glucose.
Pour over apple and bake in oven ½ hour.

FRENCH PANCAKES

INGREDIENTS ¼ oz. butter or vitaminized margarine.
½ oz. flour.
½ egg.
Saccharin.
Lemon juice.
¼ oz. 20 per cent cream.

METHOD To beaten egg add cream, flour and saccharin dissolved in a little water.
Beat mixture well.
Fry until lightly browned.
Turn and fry on other side.
Sprinkle with lemon juice and saccharin.
Fold over.

FRUIT JELLY

INGREDIENTS 5 oz. orange juice.
⅛ oz. powdered gelatine.
1 tablespoon water.
Saccharin.

Method Dissolve gelatine and saccharin in 1 tablespoon water by gently heating.
When cool, add to fruit juice.
Pour into wetted mould.

To make into orange sponge, fold in ½ egg white stiffly beaten, or whipped cream, when jelly is nearly set.

Prune Soufflé

Ingredients 3 oz. prunes (cooked).
½ egg white.
½ teaspoonful lemon juice.
Pinch salt.

Method Sieve the prunes while still warm.
Beat up the egg white to a stiff froth and fold into the above.
Add saccharin if desired dissolved in lemon juice, and a pinch of salt.
Pile up in pie-dish, and bake in oven for a few minutes.

Christmas Pudding

Ingredients ⅓ oz. breadcrumbs.
½ oz. grated carrot.
½ egg.
Pinch ground ginger.
½ teaspoon brandy.
Grated nutmeg.
⅛ oz. butter.
Rind ½ lemon.
1 oz. chopped apple.
4 oz. milk.

Method Mix all dry ingredients and pour heated milk over.
Beat up egg and add with the brandy.
Pour into buttered cup and steam for 1 to 1½ hours.

SOUPS

Clear Soup (for 2 people)

Food Value negligible.

Ingredients 1 lb. shin or shoulder of beef.
Stick of celery.
½ onion.
Salt.
½ carrot.
5 peppercorns.
1 quart water.

Method Wipe meat and cut into small pieces. Fry in a small quantity of fat till brown.
Put bones, meat and vegetables into a strong pan. Cover with water and add salt and peppercorns. Bring slowly to the boil and simmer 3 to 4 hours.
Strain. When cold, remove all fat. If desired, the stock may be clarified.
Reheat and garnish according to taste.

Cream of Celery Soup (for 2 people)

Total Food Value: Prot. 7 Fat 42 CHO 14 = Calories 462

Ingredients ½ lb. celery.
½ oz. butter or vitaminized margarine.
1 teaspoon cornflour.
½ onion.
1 pint white stock.
½ gill cream.
Salt and pepper.

METHOD Melt the butter or margarine, and sauté celery and onion.
Add the stock and cook until celery is tender (1 to 2 hours), adding more stock if necessary.
Rub through a hair sieve. Reheat.
Break down cornflour, add to soup and boil for 2 to 3 minutes.
Season and add cream.
Serve with dice of fried bread.

POTATO SOUP (for 2 people)

High carbohydrate, low fat, low protein soup.

TOTAL FOOD VALUE: Prot. 4 Fat — CHO 40 = Calories 176

INGREDIENTS

½ lb. potatoes.	2 sticks of celery.
Piece of onion.	Salt and pepper.
1 pint vegetable stock.	Pinch of herbs.

METHOD Slice the vegetables, put into pan with stock and bring to boil.
Simmer 1 to 1½ hours, adding more stock if necessary.
Rub through a hair sieve.
Reheat. Season with pepper and salt and a pinch of herbs.

TABLES *

TABLE 16

STANDARD WEIGHTS ACCORDING TO HEIGHT AND AGE PER

1. *Men.*

Age period	15 to 19	20 to 24	25 to 29	30 to 34	35 to 40	41 to 44	45 to 49	50 to 54	55
Height	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St
Ft. In.									
4 11	7 13	8 5	8 10	8 13	9 1	9 4	9 6	9 7	
5 0	8 1	8 7	8 12	9 1	9 3	9 6	9 8	9 9	
5 1	8 3	8 9	9 0	9 3	9 5	9 8	9 10	9 11	
5 2	8 6	8 12	9 2	9 5	9 7	9 10	9 12	9 13	1
5 3	8 9	9 1	9 5	9 8	9 10	9 13	10 1	10 2	1
5 4	8 12	9 5	9 8	9 11	10 0	10 2	10 4	10 5	1
5 5	9 2	9 9	9 12	10 1	10 4	10 6	10 8	10 9	1
5 6	9 6	9 13	10 2	10 5	10 8	10 10	10 12	10 13	1
5 7	9 10	10 2	10 6	10 9	10 12	11 0	11 2	11 3	1
5 8	10 0	10 6	10 10	11 0	11 3	11 5	11 7	11 8	1
5 9	10 4	10 10	11 0	11 4	11 8	11 10	11 12	11 13	1
5 10	10 8	11 0	11 4	11 9	11 13	12 1	12 3	12 4	1
5 11	10 13	11 4	11 9	12 0	12 4	12 7	12 9	12 10	1
6 0	11 4	11 9	12 1	12 6	12 10	12 13	13 1	13 2	1
6 1	11 9	12 0	12 7	12 12	13 2	13 5	13 8	13 9	1
6 2	12 0	12 5	12 13	13 4	13 9	13 12	14 1	14 2	1
6 3	12 5	12 10	13 5	13 10	14 1	14 5	14 8	14 9	1

2. *Women.*

Age period	15 to 19	20 to 24	25 to 29	30 to 34	35 to 39	40 to 44	45 to 49	50 to 54	55
Height	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	St. Lb.	S
Ft. In.									
4 11	7 12	8 1	8 4	8 7	8 10	9 0	9 3	9 5	
5 0	8 0	8 3	8 6	8 9	8 12	9 2	9 5	9 7	
5 1	8 2	8 5	8 8	8 11	9 0	9 4	9 7	9 9	
5 2	8 5	8 8	8 10	8 13	9 3	9 7	9 10	9 12	1
5 3	8 8	8 11	8 13	9 2	9 6	9 10	9 13	10 1	1
5 4	8 11	9 0	9 3	9 6	9 10	9 13	10 2	10 4	1
5 5	9 0	9 3	9 6	9 10	10 0	10 3	10 6	10 8	1
5 6	9 4	9 7	9 10	10 0	10 4	10 7	10 11	10 12	1
5 7	9 8	9 11	10 0	10 4	10 8	10 11	11 1	11 3	1
5 8	9 12	10 1	10 4	10 8	10 12	11 1	11 5	11 8	1
5 9	10 1	10 5	10 8	10 12	11 2	11 5	11 9	11 12	1
5 10	10 5	10 9	10 12	11 1	11 5	11 8	11 12	12 2	1
5 11	10 10	10 13	11 1	11 4	11 8	11 12	12 2	12 6	1
6 0	11 1	11 3	11 5	11 8	11 11	12 1	12 5	12 9	1

HEIGHT: without shoes.
WEIGHT: indoor clothes without shoes.
For naked weight subtract 10 lb. for men and 6 l
women.

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* Tables 1 to 15 appear in their appropriate places in the text.

Foodstuff	Description	Percentage of inedible waste	Protein, grams	Fat, grams	Carbohydrate, grams	Calories	Calcium, mg.	Iron, mg.	Sodium Chloride, mg.	Vitamin A, Int. units	Vitamin B₁, Int. units	Riboflavin, mg	Nicotinic Acid, mg	Ascorbic Acid, mg.	Vitamin D, Int. units
1. *Cereals*															
Arrowroot	Raw	0	0·1	0	23·0	95	3	0·3	5	0	0	—	—	0	0
Atta	Whole wheat flour	0	3·4	0·5	20·5	100	11	2·1	5	0	31	(0·06)	(0·8)	0	0
Barley, pearl	Raw	0	2·2	0·5	20·8	97	3	0·2	—	0	11	0·01	0·7	0	0
Barley, pearl	Boiled	0	0·7	0·2	7·0	33	1	0·1	—	0	(4)	Trace	(0·2)	0	0
Biscuit	Plain (water)	0	3·4	0·9	21·3	107	23	0·6	120	—	12	(0·03)	(0·3)	0	0
Biscuit	Digestive (wholemeal)	0	2·7	5·8	16·8	139	12	0·4	200	0	15	(0·03)	(0·3)	0	0
Biscuit	Sweet	0	2·0	6·8	16·8	136	14	0·3	160	0	6	(0·01)	(0·2)	0	0
Bran		0	3·1	1·2	15·5	85	28	3·7	(40)	0	65	—	1·4	0	0
Bread, white	70 per cent extraction	0	2·3	0·2	15·0	73	4	0·2	310	0	5	0·01	0·2	0	0
Bread, National	80 per cent extraction	0	2·4	0·3	14·6	70	4 (17†)	0·3	(260)	0	14	0·02	0·5	0	0
Bread, National	85 per cent extraction	0	2·4	0·4	14·6	72	4 (16†)	0·5	260	0	18	0·03	0·5	0	0
Bread, wholemeal	92 per cent extraction	0	3·1	0·6	11·2	65	7	0·7	230	0	29	0·03	0·6	0	0
Cornflakes	Breakfast cereal	0	2·1	0·1	24·3	100	3	0·8	730	0	0	(0·02)	(0·3)	0	0
Cornflour	Raw	0	0·2	0·1	24·4	99	11	0·1	35	0	0	(0·02)	(0·3)	0	0
Flour, white	70 per cent extraction	0	3·3	0·3	21·0	98	5	0·4	—	0	8	0·01	0·3	0	0
Flour, National	80 per cent extraction	0	3·4	0·4	20·2	98	5 (23†)	0·5	(5)	0	23	0·02	0·4	0	0
Flour, National	85 per cent extraction	0	3·3	0·5	20·2	99	6 (23†)	0·7	5	0	28	0·04	0·4	0	0
Flour, wholemeal	92 per cent extraction	0	4·3	0·9	17·4	95	10	1·0	5	0	43	0·06	0·8	0	0
Macaroni or spaghetti	Raw	0	3·0	0·6	19·7	96	7	0·4	5	0	7	(0·02)	(0·5)	0	0
Macaroni or spaghetti	Boiled	0	0·9	0·2	6·0	29	2	0·1	5	0	(2)	(0·01)	(0·1)	0	0
Maize meal		0	2·2	0·1	21·2	95	3	1·0	35	130(c) (yellow) 0 (white)	34	0·03	0·5	0	0
Millet finger (Ragi)	Wholegrain flour	0	2·0	0·4	21·7	98	94	1·5	[illegible]	80	[illegible]	0·33	0·4	0	0
Oatmeal	Raw	0	3·4	2·5	18·0	111	[illegible]	0·2		[illegible]	[illegible]	[illegible]			0
Oatmeal porridge	Boiled, unsalted	0	0·4	0·3	2·1	[illegible]	20	0·1	—	(15)(c)	(1)	(0·01)	(0·1)	2	0
Rice, milled (polished rice)	Raw	0	1·8	0·3	22·9	1	20	0·1	—	6(c)	2	—	—	2	0
Rice, milled (polished rice)	Boiled	0	0·5	0·1	1·6	7	6	0·2	—	(3)(c)	1	—	—	1	0
Rice, unmilled (brown rice)	Raw	0	2·0	[illegible]	2·0	9	3	0·1	—	4(c)	2	0·01	0·1	17	0
Rye	Wholegrain flour	0	2·4						—	60(c)	3	0·01	0·1	4	0
Ryvita		0	0·1	0	4·1	17	8	0·9							
Sago	Raw	0	0·7	0	12·8	53	20	[illegible]	—	72(c)	9	0·01	0·1	1	0
Semolina	Raw								15	330(c)	0	0·02	0·3	0	0
Sorghum or Cholam (Kaffir corn, Indian millet or Jowar)	Wholegrain flour	—	0·1	0	1·1	5	1	0·1	—	23(c)	2	—	—	3	0
Tapioca	Raw														
Wheatflakes, shredded wheat, etc	Breakfast cereal														

(c) The sole source of vitamin A potency is carotene.

* For instructions on

Foodstuff	Description	Percent of inedi waste	Pro gra												
2. Dairy Products															
Butter		0	0·1	23·4	0	211	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Cheese	Cheddar type	5	7·1	9·8	0	117	230	0·2	[illegible]	[illegible]	[illegible]	[illegible]	0·1	0	10
Cream	40 per cent	0	0·5	11·9	0·6	111	17	0·1	25	190	3	(0·01)	0	0	(4)
Egg, fresh *	Raw or boiled	12†	3·5	3·3	0·3	45	17	0·8	75	280	14	0·11	Trace	0	17
Egg, fresh *	Fried	0	4·1	5·5	0·3	67	18	0·7	90	(330)	(13)	(0·10)	Trace	0	(20)
Egg, fresh *	Poached	0	3·6	3·3	0·3	45	15	0·6	75	280	(11)	(0·08)	Trace	0	17
Egg	Dehydrated	0	13·0	11·9	0·9	163	62	3·1	(280)	850	38	0·37	0·1	0	68
Ghee	Clarified butterfat	0	0	26·0	0	228	0	0	—	590	0	0	0	0	12
Milk, whole	Fresh	0	0·9	1·0	1·2	17	34	Trace	35	30	4	0·04	Trace	0·3	0·3
Milk, whole, condensed	Unsweetened (U.K.)	0	2·4	2·6	3·3	46	83	0·1	120	105	6	0·10	0·1	1	1
Milk, whole, condensed	Unsweetened (U.S.A.), (evaporated)	0	2·0	2·3	2·7	40	69	0·1	(100)	90	5	0·10	(0·1)	1	1
Milk, whole, condensed	Sweetened	0	2·3	2·6	14·1	89	82	0·1	100	105	10	0·10	(0·1)	1	1
Milk, whole	Dried	0	7·3	7·6	10·1	138	254	0·2	290	300	28	0·33	0·2	0	3
Milk, skimmed	Fresh	0	1·0	0	1·3	9	35	Trace	40	0	4	0·05	Trace	0·4	0
Milk, skimmed	Dried	0	10·2	0·2	13·6	97	348	0·3	(310)	9	37	0·45	0·3	0	0
3. Meat and Poultry															
Bacon	Home-cured raw	12	3·1	12·8	0	128	3	0·3	810	0	56	0·06	0·3	0	0
Bacon	Fried	0	7·6	11·9	0	137	8	0·9	1,770	0	42	0·05	0·2	0	0
Beef	Raw – average of good quality meat	17	4·2	8·0	0	89	3	1·1	30	14	7	0·07	1·3	0	0
Beef	Raw – low grade lean or native	25	4·8	4·5	0	60	3	1·1	30	14	7	0·07	1·3	0	0
Beef	Boiled (edible portion only)	—	9·5	2·3	0	61	1	2·4	20	(7)	3	0·06	1·0	0	0
Beef	Roast, lean and fat (edible portion only)	—	6·4	7·9	0	97	2	1·3	30	(14)	4	0·06	1·0	0	0
Beef	Roast, lean only (edible portion only)	—	7·6	3·9	0	65	2	1·4	30	(7)	4	0·06	1·0	0	0
Rumpsteak	Grilled (edible portion only)	—	7·2	6·1	0	84	3	1·5	30	(10)	4	0·06	1·0	0	0
Beef	Canned, corned	0	7·1	4·5	0	69	3	3·1	920	0	0	(0·04)	(0·5)	0	0
Beef	Dehydrated	0	14·2	11·4	0	159	4	3·4	95	43	11	0·40	4·0	0	0
Duck	Raw, plucked and drawn	30	5·4	2·3	0	42	3	0·6	20	0	14	(0·03)	(2·3)	0	0
Duck	Roast (edible portion only)	—	6·5	6·7	0	88	5	1·6	75	0	8	(0·02)	(1·8)	0	0
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	0	[illegible]	[illegible]	[illegible]	10	0	14	0·03	2·3	0	0

* Average egg weighs about 2 oz.

† Shell.

STANDARD

1. *Men.*

Age period	15 to 19	
Height	St. Lb.	
Ft. In.		
4 11	7 13	
5 0	8 1	
5 1	8 3	
5 2	8 6	
5 3	8 9	
5 4	8 12	
5 5	9 2	
5 6	9 6	
5 7	9 10	
5 8	10 0	
5 9	10 4	
5 10	10 8	
5 11	10 13	
6 0	11 4	
6 1	11 9	
6 2	12	
6 3	12	

2. *W*

Age period	
Height	
Ft. I	
4	

Calories	Calcium, mg.	Iron, mg.	Sodium Chloride, mg.	Vitamin A, Int. units	Vitamin B_1, Int. units	Riboflavin, mg.
55	8	0·2	30	42		
		0·4	400	71		
				77		

Heart		[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Kidney			·5	2·0	0	36	3	3·8	120	280	22	0·37	3·8	0	0
Kidney			·3	1·6	0	44	6	2·0	70	(280)	13	0·24	1·9	0	0
Liver			·8	1·7	1·4	40	3	3·9	50	4,260	38	0·85	3·8	0	0
Liver			·4	4·5	1·0	78	2	5·9	40	6,050	42	0·68	2·7	0	0
Luncheon me			·3	6·0	0	71	3	0·6	(980)	0	42	(0·04)	(0·7)	0	0
Meat paste			·4	2·3	2·6	45	7	1·0	(690)	0	4	(0·03)	(0·6)	0	0
Meat roll			·1	6·8	0·8	90	16	0·9	(620)	0	(10)	(0·04)	(0·6)	0	0
Mutton		17	3·7	8·8	0	94	3	0·6	35	14	15	0·05	1·2	0	0
Mutton	u only)	—	7·3	4·7	0	71	1	1·4	30	(7)	6	0·04	1·0	0	0
Mutton	only)	—	7·1	5·8	0	81	1	1·2	30	(9)	14	0·04	1·0	0	0
Mutton chop		22	4·4	17·1	0	171	4	0·7	45	(14)	11	0·04	0·8	0	0
Mutton chop		44	5·7	12·8	0	138	5	0·7	40	(20)	10	0·04	1·0	0	0
Pork		15	3·4	11·4	0	116	3	0·3	40	0	67	(0·06)	1·7	0	0
Pork	Roast (edible p… only)	—	6·2	9·0	0	106	2	0·6	40	0	90	(0·05)	1·4	0	0
Pork chop	Grilled	40	5·3	14·3	0	150	2	0·7	35	0	43	(0·05)	1·4	0	0
Pork sausage meat	Canned	0	2·6	12·2	0·3	121	3	0·6	(980)	0	42	(0·04)	(0·7)	0	0
Rabbit	Skinned and gutted, raw	17	5·7	1·7	0	38	3	0·6	70	0	3	0·14	1·8	0	0
Rabbit	Stewed	(17)	7·6	2·2	0	50	3	0·5	20	0	1	0·09	0·9	0	0
Sausages	Beef, raw	0	3·3	3·7	3·7	61	9	0·8	500	3	14	(0·02)	(0·5)	0	0
Sausages	Beef, fried	0	3·9	5·2	4·5	80	6	1·1	800	(3)	10	(0·02)	(0·3)	0	0
Sausages	Pork, raw	0	3·0	5·1	3·7	73	9	0·3	500	0	16	(0·02)	(0·5)	0	0
Sausages	Pork, fried	0	3·3	7·1	3·6	91	6	0·9	700	0	12	(0·02)	(0·5)	0	0
Sausage, breakfast	Cooked	0	3·0	4·0	7·2	80	6	0·5	600	0	(3)	(0·04)	(0·7)	0	0
Sweetbreads	Raw	40	6·2	4·5	0	68	18	0·4	30	(10)	30	(0·03)	(1·0)	0	0
Sweetbreads	Stewed (edible portion only)	—	6·4	2·6	0	49	4	0·5	35	(6)	12	(0·02)	0·5	0	0
Tongue	Canned	0	5·4	5·7	0	73	3	0·9	1,380	0	26	(0·03)	3·6	0	0
Tongue	Stewed (edible portion only)	—	5·1	6·8	0	82	3	1·0	40	0	6	(0·02)	1·8	0	0
Tripe	Raw	45	3·3	0·3	0	17	20	0·2	15	(10)	(16)	0·03	(1·0)	0	0
Tripe	Stewed (edible portion only)	—	5·1	0·9	0	28	36	0·4	15	(10)	(6)	0·02	(0·5)	0	0
Veal	Raw	27	5·1	1·7	0	36	3	0·6	30	14	7	0·04	2·0	0	0
Veal, fillet	Roast (edible portion only)	—	8·7	3·3	0	64	4	1·2	50	(28)	(7)	0·03	1·6	0	0
4. *Fish*															
Fish, white	Fresh, raw	45	4·5	0·3	0	21	7	0·3	50	0	6	0·04	0·6	0	0
Fish, white	Steamed (edible portion only)	—	5·5	0·5	0	26	11	0·2	55	0	4	0·03	0·4	0	0
Fish, white	Fried in batter	—	5·3	3·4	1·4	57	24	0·8	80	0	5	0·03	0·4	0	0
Fish, white	Hard dried	35	11·9	0·4	0	[illegible]	[illegible]	0·2	—	(15)(c)	(1)	(0·01)	(0·1)	2	0
Fish paste	Canned	0	3·8	2·5	[illegible]	2	29	0·1	—	6(c)	2	—	—	3	0
Haddock	Smoked	45	5·1	[illegible]	0·2	1	20	0·1	—	(3)(c)	1	—	—	1	0
Herring	Fresh, raw	30	[illegible]	0	1·6	7	6	0·2	—	4(c)	2	0·01	0·1	17	0
Herring	Baked in vinegar (edible portion only)	—	0·2	0	2·0	9	3	0·1	—	60(c)	3	0·01	0·1	4	0
Herring	Fried in oatmeal (edib'	0	0·1	0	4·1	17	8	0·2	—	72(c)	2	0·01	0·1	1	0
	portion only)	—	0·7	0	12·5	53	26	0·7	15	350(c)	0	0·02	0·3	0	0
Herring	Kippered														
Herring	Canned in tomato ', aver-	—	0·1	0	1·1	5	1	0·1	—	25(c)	2	—	—	3	0
Pilchards	Canned in tomaallowance														
Roe	Cod, raw														
Roe	Cod, fried ir														
Salmon	Fresh, raw														

(c) The sole source of vitamin A potency is carotene.

STANDARD

1. *Men.*

Age period	15 to 19
Height	St. Lb.
Ft. In.	
4 11	7 13
5 0	8 1
5 1	8 3
5 2	8 6
5 3	8 9
5 4	8 12
5 5	9 2
5 6	9 6
5 7	9 10
5 8	10 0
5 9	10 4
5 10	10 8
5 11	10 13
6 0	11 4
6 1	11 [illegible]
6 2	12
6 3	12

2. *W*

Age period	
Heigh	
Ft.	
4	

Foodstuff	Descript	aste	Pro												
Salmon	Fresh, steamed (edible portion only)	—	5·4	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Salmon	Canned	0	5·7	2·8	0	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Sardines	Canned in oil	0	5·7	6·8	0	84	114	1·1	580	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	280
Shellfish, crab or lobster	With shells, raw	60	5·7	1·1	0·3	34	13	0·3	250	0	11	0·07	2·5	0	0
Shellfish, crab or lobster	Boiled (edible portion only)	—	5·7	1·1	0	33	12	0·3	260	0	5	0·06	1·7	0	0
Shellfish, oysters, cockles, mussels	With shells, raw	80	3·1	0·4	1·3	22	38	3·6	290	44	16	0·13	5·0	0	1
5. Fats and Oils															
Dripping		0	0	28·1	0	253	0	0	—	28	0	0	0	0	8
Lard		0	0	28·1	0	253	0	0	—	0	0	0	0	0	0
Margarine	Vitaminized	0	0	24·2	0	218	1	0·1	230	570	0	0	0	0	56
Oil, vegetable	Cooking	0	0	28·4	0	256	0	0	—	0	0	0	0	0	0
Red palm oil		0	0	28·4	0	256	0	0	—	12,000(c)–30,000(c)	0	0	0	0	0
6. Fruits															
Apple	English, eating	20	0·1	0	3·0	12	1	0·1	—	11(c)	4	Trace	0·1	1	0
Apple	Dried, raw	0	0·6	0	12·5	52	8	0·6	10	28(c)	0	(0·01)	(0·4)	0	0
Apple	Baked	30	0·1	0	2·5	10	1	0·1	—	11(c)	3	Trace	0·1	0	0
Apple	Stewed without sugar (edible portion only)	—	Trace	0	1·1	4	Trace	Trace	—	9(c)	3	Trace	0·1	0	0
Apricot	Fresh	8	0·2	0	2·0	7	5	0·1	—	213(c)	3	0·02	(0·1)	3	0
Apricot	Dried, raw	0	1·4	0	11·1	50	26	1·2	15	1,420(c)	0	(0·12)	(0·6)	0	0
Apricot	Dried, stewed without sugar	0	0·6	0	4·6	21	26	1·2	5	486(c)	0	(0·05)	(0·2)	0	0
Apricot	Canned	0	0·1	0	3·5	14	4	0·1	—	142(c)	2	(0·02)	(0·1)	0	0
Avocado pear	Raw	(40)	0·5	6·5	1·6	68	3	0·2	—	54(c)	14	(0·03)	(0·3)	1	0
Banana	Raw	40	0·3	0	4·9	21	2	0·1	—	23(c)	5	0·01	0·2	3	0
Blackberries	Raw	0	0·4	0	1·6	8	18	0·3	5	85(c)	2	(0·01)	(0·1)	6	0
Cape gooseberry	Raw	0	0·5	0·1	3·3	16	3	0·5	—	—	—	(0·01)	(0·1)	14	0
Cherry	Raw	13	0·2	0	3·0	13	4	0·1	0	57(c)	5	(0·01)	(0·1)	1	0
Cherry	Stewed without sugar (weighed with stones)	5	0·1	0	1·1	5	2	Trace	0	27(c)	3	—	—	1	0
Currants, black	Raw	0	0·3	0	1·7	8	17	0·4	0	26(c)	4	(0·01)	(0·1)	57	0
Currants, black	Stewed without sugar	0	0·2	0	1·2	6	12	0·2	0	18(c)	2	(0·01)	(0·1)	46	0
Currants, red	Raw	0	0·3	0	1·1	6	10	0·3	0	7(c)	4	(0·01)	(0·1)	13	0
Currants, red	Stewed without sugar	0	0·2	0	0·8	4	7	0·2	0	7(c)	2	—	—	8	0
Damsons	Raw	10	0·1	0	2·4	10	7	0·1	0	—	5	(0·01)	(0·1)	1	0
											8	—	—	1	0

Figs	[illegible], stewed without sugar	0	0·6	0	7·6	33	40	0·7	35	15(c)	0	0·05	0·3	0	0
Gooseberry	Eating, raw	0	0·2	0	2·4	10	5	0·2	—	57(c)	4	(0·01)	(0·1)	11	0
Gooseberry	Stewed without sugar	0	0·2	0	0·4	2	4	Trace	0	57(c)	2	—	—	7	0
Grape, green	Raw	10	0·2	0	4·1	17	5	0·1	Trace	14(c)	3	(0·01)	(0·1)	1	0
Grapefruit	Raw	50	0·2	0	1·4	6	5	0·1	—	6(c)	7	(0·01)	(0·1)	14	0
Grapefruit	Canned	0	0·1	0	3·6	15	5	0·1	—	4(c)	4	(0·01)	(0·1)	7	0
Greengage	Raw	6	0·2	0	3·0	13	5	0·1	—	114(c)	4	(0·01)	(0·1)	1	0
Greengage	Stewed without sugar (weighed with stones)	4	0·1	0	2·0	8	3	0·1	—	(60)(c)	2	—	—	1	0
Guava pear		(25)	0·4	0·1	4·1	19	3	0·3	20	42(c)	11	0·01	0·3	52	0
Lemon		30	0·2	0	0·8	5	20	0·1	—	0	3	0	0	12	0
Lime		(80)	0·4	0	3·1	14	26	0·6	—	12(c)	(3)	0	0	11	0
Loganberry	Raw	0	0·3	0	0·9	5	10	0·4	—	20(c)	2	—	—	9	0
Mango	Ripe	(40)	0·2	0	4·0	16	2	0·1	10	1,260(c)	6	0·02	(0·1)	12	0
Melon	Yellow	45	0·2	0	1·3	6	4	0·1	—	24(c)	4	0·01	0·2	3	0
Orange	Sweet	25	0·2	0	2·2	10	12	0·1	—	85(c)	7	0·01	0·1	16	0
Orange juice		0	0·2	0	2·4	10	3	0·1	—	36(c)	3	—	—	10	0
Papaya (paw paw)	Ripe	(45)	0·1	0	2·7	11	3	0·1	—	960(c)	2	0·05	(0·1)	13	0
Peach	Fresh, raw	13	0·2	0	2·3	10	1	0·1	—	213(c)	2	0·01	0·2	3	0
Peach	Dried, raw	0	1·0	0	13·5	58	10	1·9	—	(1,050)(c)	0	(0·05)	(1·0)	0	0
Peach	Dried, stewed without sugar	0	0·3	0	4·6	20	3	0·6	—	(320)(c)	0	(0·01)	(0·3)	0	0
Peach	Canned	0	0·1	0	3·9	16	2	0·2	—	142(c)	1	0	(0·1)	1	0
Pear	Eating, raw	25	0·1	0	2·7	11	2	0·1	—	3(c)	2	0·01	0·1	1	0
Pear	Cooking, stewed without sugar	0	0·1	0	1·6	7	1	Trace	—	0	1	0	0	0	0
Pear	Canned	0	0·1	0	3·6	15	3	0·2	—	0	1	0	0	0	0
Pineapple	Fresh	50	0·1	0	3·0	12	3	0·1	—	28(c)	7	(0·02)	(0·2)	6	0
Pineapple	Canned	0	0·1	0	4·9	20	3	0·2	—	18(c)	5	(0·01)	(0·1)	3	0
Plantain		(40)	0·3	0	7·0	30	3	0·1	—	255(c)	(5)	(0·01)	(0·2)	1	0
Plum	Dessert, raw	6	0·2	0	1·6	7	4	0·1	—	114(c)	4	0·01	0·2	1	0
Plum	Cooking, stewed without sugar (weighed with stones)	4	0·1	0	1·0	4	2	Trace	0	(60)(c)	(2)	0	(0·1)	0	0
Plum	Canned	0	0·1	0	4·6	19	3	0·4	—	57(c)	2	0	(0·1)	1	0
Prune	Dried, raw	17	0·7	0	10·3	44	11	0·8	—	710(c)	0	0·04	0·6	0	0
Prune	Dried, stewed without sugar (weighed with stones)	8	0·3	0	4·0	17	4	0·3	—	(806)(c)	0	(0·02)	(0·3)	0	0
Raisin	Dried	8	0·3	0	16·5	67	17	0·5	5	14(c)	0	0·01	0·1	0	0
Raspberry	Raw	0	0·3	0	1·4	7	12	0·3	—	20(c)	2	(0·01)	(0·2)	9	0
Raspberry	Stewed without sugar	0	0·2	0	1·0	5	8	0·2	—	(15)(c)	(1)	(0·01)	(0·1)	2	0
Rhubarb	Raw	25	0·2	0	0·3	2	29	0·1	—	6(c)	2	—	—	3	0
Rhubarb	Stewed without sugar	0	0·1	0	0·2	1	20	0·1	—	(3)(c)	1	—	—	1	0
Strawberry	Raw	3	0·2	0	1·6	7	6	0·2	—	4(c)	2	0·01	0·1	17	0
Fruit, dessert, fresh	Average of 12 (waste allowance made)	—	0·2	0	2·0	9	3	0·1	—	60(c)	3	0·01	0·1	4	0
Fruit, canned	Average of 5	0	0·1	0	4·1	17	3	0·2	—	72(c)	2	0·01	0·1	1	0
Fruit, dried	Average of 6 (waste allowance made)	—	0·7	0	12·5	53	26	0·7	15	350(c)	0	0·02	0·3	0	0
Fruit, fresh	Stewed without sugar, average of 11 (waste allowance made)	—	0·1	0	1·1	5	1	0·1	—	25(c)	2	—	—	3	0

(c) The sole source of vitamin A potency is carotene.

Foodstuff	Description	Percentage of inedible waste	Protein, grams	Fat, grams	Carbohydrate, grams	Calories	Calcium mg.	Iron, mg.	Sodium Chloride, mg.	Vitamin A, Int. units	Vitamin B_1, Int. units	Riboflavin, mg.	Nicotinic Acid, mg.	Ascorbic Acid, mg.	Vitamin D, Int. units
7. *Nuts and Oil Seeds*															
Almond		65	5·8	15·2	1·1	164	70	1·2	—	0	22	(0·05)	0·4	0	0
Brazil nut		55	3·9	17·5	1·0	177	50	0·8	—	0	100	(0·05)	(0·4)	0	0
Chestnut		20	0·6	0·8	9·3	47	13	0·3	5	0	19	(0·05)	0·4	0	0
Cobnut, hazel nut		65	2·6	10·2	1·7	109	12	0·3	—	0	58	(0·05)	0·4	0	0
Gingelly seeds, simsin, beniseed		—	5·2	12·3	7·1	160	412	3·0	—	9	—	—	—	0	0
Peanut, ground nut		30	8·0	13·9	2·2	166	17	0·7	5	0	84	0·08	0·6	0	0
Pecan		(60)	2·7	20·0	4·3	216	25	0·7	—	110(c)	57	0·08	(0·4)	0	0
Walnut	With shells, dried	40	3·5	14·6	1·3	151	17	0·7	—	0	28	(0·05)	(0·4)	0	0
8. *Vegetables*															
Amaranth	Tender	—	1·4	0·1	1·6	13	14	6·1	—	3,600(c)	3	0·03	(0·1)	24	0
Asparagus	Raw	80	0·6	0	0·7	5	8	0·3	—	1,200(c)	17	0·03	0·2	17	0
Asparagus	Boiled	80	1·0	0	0·3	5	7	0·2	—	(246)(c)	21	0·05	0·3	17	0
Beans, broad	Fresh, raw	75 (pods)	2·0	0·1	2·7	20	8	0·3	—	—	(18)	(0·04)	(0·3)	8	0
Beans, broad	Boiled	0	1·2	0	1·8	12	6	0·3	5	—	(18)	(0·03)	(0·1)	2	0
Beans, French	Raw	5	0·3	0	0·7	4	9	0·2	10	170(c)	7	0·03	(0·1)	3	0
Beans, French	Boiled	0	0·2	0	0·3	2	11	0·2	5	(84)(c)	3	0·01	(0·1)	2	0
Beans, runner	Raw	25	0·3	0	0·7	4	9	0·2	(10)	170(c)	2	0·02	(0·2)	6	0
Beans, runner	Boiled	0	0·2	0	0·3	2	7	0·2	5	(84)(c)	1	0·01	(0·1)	2	0
Beans, stringless and runner	Canned	0	0·2	0	0·5	3	10	0·1	—	170(c)	2	(0·02)	(0·2)	1	0
Beans, baked in tomato sauce	Canned	0	1·7	0	4·5	25	16	0·7	400	142(c)	6	0·01	0·2	1	0
Beans, haricot	Dried	0	6·1	0	11·6	71	51	1·9	—	0	42	0·08	0·6	0	0
Beans, haricot	Boiled	0	1·9	0	4·2	24	18	0·7	—	0	10	0·02	0·1	0	0
Beans, soya	Dried, whole	0	8·5	4·3	9·4	114	67	2·6	—	75(c)	80	0·06	1·2	0	0
Beans, soya	Flour, full fat	0	11·5	6·7	3·8	122	62	2·0	—	0	62	0·04	0·9	0	0
Beetroot	Raw	35	0·5	0	2·3	11	9	0·3	30	0	3	0·02	0·2	3	0
Beetroot	Boiled	0	0·5	0	2·5	12	8	0·2	35	0	3	0·02	0·1	2	0
Brussels sprouts	Raw	25	1·2	0	1·1	10	8	0·3	(15)	114(c)	11	0·02	0·1	28	0
Brussels sprouts	Boiled	0	0·7	0	0·4	4	8	0·2	5	(45)(c)	6	0·01	0·1	9	0
Brussels sprouts	Canned	0	1·0	0	0·4	6	9	0·2	—	114(c)	5	(0·02)	(0·1)	10	0
Cabbage	Raw	30	0·4	0	1·4	7	18	0·3	10	256(c)	7	0·02	0·1	20	0
Cabbage	Boiled	0	0·2	0	0·3	2	12	0·1	5	90(c)	2	0·01	Trace	11	0
Cabbage	Dehydrated	0	5·3	0·4	15·2	88	188	0·3	170	3,400(c)	0	(0·15)	(0·7)	90	0
Cabbage, Chinese	Raw	(30)	0·4	0	0·7	5	(18)	0·2	—	2,700(c)	25	(0·01)	(0·1)	11	0
Cabbage, red	Raw	30	0·6	0	1·6	9	5	0·2	20	0	(7)	(0·02)	(0·1)	16	0
Carrot	Raw	5 new	0·2	0	1·4	6	14	0·2	80	4,250(c)	6	0·01	0·2	8	0

Carrot	Dehydrated														
Cassava or manioc	Fresh tuber	—	0·8	0·1	8·5	36	8	0·2	—	0	0	0·13	—	8	0
Cassava gari	Flour from soaked tuber	—	0·2	0	23·3	96	15	0·1	0	0	0	0·07	—	0	0
Cauliflower	Raw	30	0·7	0	0·8	6	14	0·2	(10)	0	9	0·02	0·2	20	0
Cauliflower	Boiled	0	0·4	0	0·3	3	6	0·1	5	0	2	0·01	0·1	15	0
Celery	Raw	25	0·3	0	0·3	2	15	0·2	75	0	8	0·01	0·1	Trace	0
Celery	Boiled	0	0·2	0	0·2	1	15	0·1	50	0	1	0·01	0·1	0	0
Colocasia	Raw	(25)	0·8	0	6·8	29	11	0·6	—	18(c)	23	—	—	Trace	0
Cucumber	Raw	25	0·2	0	0·4	3	6	0·1	10	0	4	Trace	0·1	3	0
Dhal arhar	Red gram, raw	0	6·3	0·5	16·3	95	40	2·5	(15)	105(c)	42	0·02	1·5	0	0
Egg plant (Brinjal)	Raw	20	0·3	0·1	1·3	7	3	0·2	—	24(c)	6	0·02	0·2	2	0
Endive	Raw	35	0·5	0	0·3	3	12	0·8	10	2,010(c)	6	—	—	6	0
Gourd, bitter	Raw	—	0·5	0·1	1·2	7	6	0·6	—	110(c)	5	0·03	—	16	0
Gourd, snake	Raw	—	0·1	0·1	1·2	6	14	0·4	5	340(c)	(5)	0·02	—	34	0
Horseradish	Raw	55	1·3	0	2·8	16	34	0·6	—	0	8	0·01	—	37	0
Ipomoea	Leaves, raw	—	0·8	0·1	1·2	19	31	1·1	—	1,900(c)	8	0·03	—	23	0
Kale	Raw	30	1·1	0	1·3	10	57	0·7	—	2,270(c)	11	0·01	—	37	0
Kale	Boiled	0	1·1	0	2·0	12	(57)	(0·7)	—	(2,270)(c)	7	0·01	—	9	0
Leek	Raw	50	0·7	0	1·1	7	14	0·4	(5)	200(c)	9	—	—	6	0
Leek	Boiled	0	0·5	0	1·2	7	17	0·6	5	(135)(c)	3	—	—	3	0
Lentils	Raw	0	6·8	0	13·6	82	11	2·2	25	14(c)	42	0·02	0·9	0	0
Lentils	Boiled	0	1·9	0	4·7	26	3	0·6	5	(3)(c)	6	Trace	0·2	0	0
Lettuce	Raw	20	0·3	0	0·5	3	7	0·2	—	1,140(c)	7	0·02	0·1	4	0
Marrow, vegetable	Raw	35	0·1	0	0·8	4	4	0·1	—	8(c)	4	0·01	0·2	1	0
Marrow, vegetable	Boiled	0	0·1	0	0·4	2	4	0·1	—	(5)(c)	3	0·01	0·2	1	0
Onion, bulb	Raw	5	0·3	0	1·3	6	9	0·1	5	0	3	0·01	Trace	3	0
Onion, bulb	Boiled	0	0·2	0	0·7	4	7	0·1	—	0	2	0·01	Trace	1	0
Onion, spring	Raw	20	0·3	0	2·2	10	38	0·3	—	200(c)	2	—	—	6	0
Parsley	Raw	0	1·5	0	0	6	92	2·3	—	3,690(c)	11	0·02	—	43	0
Parsnip	Raw	35	0·5	0	2·9	14	16	0·2	15	57(c)	11	0·02	—	3	0
Parsnip	Boiled	0	0·4	0	3·4	15	10	0·1	5	(54)(c)	6	0·02	—	1	0
Peas, green	Fresh, raw	60	1·6	0	2·7	17	4	0·5	10	140(c)	39	0·03	0·2	8	0
Peas, green	Fresh, boiled	0	1·4	0	2·0	14	4	0·3	—	(105)(c)	29	0·03	0·2	3	0
Peas, green	Canned	0	1·3	0	1·6	12	7	0·3	160	140(c)	23	0·03	0·2	4	0
Peas	Dried, raw	0	7·0	0	14·2	85	17	1·3	30	57(c)	42	0·08	0·6	0	0
Peas	Dried, boiled	0	2·0	0	4·9	28	7	0·4	5	(12)(c)	18	0·02	0·1	0	0
Pepper, red (chillies, pimento)	Fresh	—	0·6	0·2	2·0	12	5	0·2	5	825(c)	2	0·04	0·1	40	0
Pepper, red (chillies, pimento)	Dried	0	2·7	2·2	19·9	110	45	4·0	—	11,400(c)	—	(0·12)	0·3	140	0
Potatoes	Fresh, raw	7 early 25 old	0·6	0	4·6	21	2	0·2	5	0	11	0·02	0·3	8 early 2 old	0
Potatoes	Fresh, boiled	0	0·4	0	4·9	21	1	0·1	5	0	8	0·01	0·2	5 early 2 old	0
Potatoes	Fresh, fried (chips)	0	1·1	2·6	9·5	66	4	0·4	10	0	(8)	(0·01)	(0·2)	4	0
Potatoes	Fresh, roast	0.	0·8	0·3	7·0	34	3	0·3	5	0	7	(0·01)	(0·2)	1	0
Potatoes	Canned	0	0·5	0	4·1	18	11	0·1	—	0	6	(0·02)	(0·3)	2	0
Potatoes	Dehydrated	0	2·5	0	23·0	101	24	0·9	5	0	27	(0·10)	(1·5)	12	0
Pumpkin	Raw	20	0·2	0	1·0	6	9	0·2	—	45(c)	3	0·01	0·2	3	0
Radish	Raw	50	0·3	0	0·7	4	12	0·5	10	0	6	0·01	—	8	0
Spinach	Raw	25	0·8	0	0·7	6	20	0·9	85	2,000(c)	9	0·06	0·1	18	0
Spinach	Boiled	0	1·4	0	0·4	7	(35)	1·1	25	(2,460)(c)	10	0·08	0·1	17	0
Spinach	Canned	0	0·6	0	0·3	4	16	0·3	—	2,000(c)	3	(0·06)	0·1	7	0
Sugar cane	Raw	—	Trace	0	5·8	24	3	0·1	—	33(c)	—	—	—	Trace	0
Swedes	Raw	35	0·3	0	1·1	6	16	0·1	15	0	3	(0·01)	(0·3)	11	0
Swedes	Boiled	0	0·3	0	1·0	5	12	0·1	5	0	2	(0·01)	(0·2)	7	0
Sweet potato	Raw	12	0·4	0·2	7·7	34	8	0·4	40	1,500(c) (yellow) 0 (white)	7	0·03	0·4	7	0

(c) The sole source of vitamin A potency is carotene.

Foodstuff	Description	Percentage of inedible waste	Protein, grams	Fat, grams	Carbohydrate, grams	Calories	Calcium, mg.	Iron, mg.	Sodium Chloride, mg.	Vitamin A, Int. units	Vitamin B_1, Int. units	Riboflavin, mg.	Nicotinic Acid, mg.	Ascorbic Acid, mg.	Vitamin D, Int. units
Sweet potato	Boiled	0	0·3	0	5·1	22	6	0·2	15	840(c) (yellow) 0 (white)	5	0·03	0·3	3	0
Tomato	Raw	15	0·3	0	0·7	4	4	0·1	—	850(c)	6	0·01	0·1	7	0
Tomato	Fried	0	0·3	1·7	0·8	20	4	0·1	—	850(c)	4	0·01	0·1	4	0
Tomato	Canned	0	0·3	0	1·0	5	2	0·3	—	850(c)	4	0·01	0·1	5	0
Turnip	Raw	35	0·2	0	1·0	5	17	0·1	35	0	3	0·01	0·3	7	0
Turnip	Boiled	0	0·2	0	0·6	3	17	0·1	15	0	2	0·01	0·2	4	0
Turnip	Canned	0	0·1	0	0·5	2	16	0·1	—	0	1	(0·01)	(0·2)	1	0
Turnip tops	Raw	25	0·7	0	1·0	7	100	1·0	(10)	2,840(c)	11	(0·02)	(0·4)	28	0
Watercress	Raw	15	0·8	0	0·2	4	63	0·4	40	1,420(c)	11	(0·02)	(0·5)	17	0
Yams	Ordinary, raw	12	0·6	0	5·3	25	17	0·4	—	0	7	(0·03)	0·2	3	0
Vegetables, fresh, English type, average of 20	Raw, excluding potatoes (waste allowance made)	—	0·3	0	0·8	4	10	0·2	20	500(c)	5	0·01	0·1	7	0
Vegetables, fresh, English type, average of 18	Boiled, excluding potatoes	0	0·5	0	1·0	5	14	0·3	10	540(c)	5	0·01	0·1	5	0
Vegetables, canned, average of 7	Excluding potatoes and baked beans	0	0·5	0	0·6	5	10	0·2	(170)	1,280(c)	6	0·02	0·2	4	0
9. Sugar, Sweets and Preserves															
Chocolate	Plain	0	1·3	9·2	15·0	148	7	0·9	5	21(c)	9	0·07	0·3	0	0
Honey	Drained	0	0·1	0	19·5	78	1	0·1	—	0	0	0·02	0·1	0	0
Jaggery or gur	Crude native sugar	0	0·1	0	27·0	109	23	3·2	—	130(c)	—	—	—	0	0
Jam		0	0·1	0	17·6	71	8	0·3	—	6(c)	Trace	0	0	3	0
Jelly packet	Cubes	0	2·4	0	17·1	77	10	0·4	15	0	0	0	0	0	0
Jelly	Prepared	0	0·6	0	4·9	22	3	0·1	5	0	0	0	0	0	0
Lemon curd		0	0·3	0·9	15·9	78	1	0·1	40	13	0	0	0	0	0
Sugar, white		0	0	0	27·0	108	0	0	0	0	0	0	0	0	0
Syrup, golden		0	0·1	0	20·2	81	7	0·4	0	0	0	0	0	0	0
Sweets, boiled		0	0	0	26·2	105	0	0	0	0	0	0	0	0	0
Toffee		0	0·9	5·7	18·5	129	28	0	—	28	—	—	—	0	—
Treacle, black		0	0·3	0	17·2	70	141	2·6	70	0	0	0·02	1·1	0	0
10. Beverages															
Cocoa	Powder	0	5·8	7·3	8·9	125	14	4·1	95	43(c)	11	0·08	(0·3)	0	0
Cocoa, beverage as drunk	With milk and sugar	0	0·2	0·2	0·9	6	3	Trace	10	8	Trace	Trace	Trace	0	0
Marmite		0	2·8	0	0	11	22	1·4	4,000	0	140	1·70	19·0	0	0
Meat extract		0	8·6	0·6	0	41	22	3·7	5,000	0	25	0·48	17·0	0	0
Ovaltine	Powder	0	3·9	2·2	18·1	108	100	1·0	180	114	37	0·17	—	0	0

Ale, strong					as alcohol)										
Beer		0	0·1	0	0·5 (+1·0 as alcohol)	10	8	0	—	0	Trace	0·01	(0·4)	0	0
Liqueur		0	0	0	8·2 (+8·2 as alcohol	95	—	—	—	0	0	0	0	0	0
Stout		0	0·1	0	1·0 (+1·2 as alcohol)	13	1	0	—	0	Trace	(0·01)	(0·4)	0	0
Whisky or brandy		0	0	0	(9·9 as alcohol)	70	0	0	—	0	0	0	0	0	0
Wine	Light table	0	0·1	0	0·3 (+2·8 as alcohol)	20	—	—	—	0	—	—	—	0	0
Wine, port or sherry	Dessert, fortified	0	0·1	0	1·3 (+4·2 as alcohol)	35	—	—	—	0	—	—	—	0	0
Toddy	Coconut (fermented)	0	Trace	0·1	0·5	3	3	0·8	—	—	—	—	—	—	—
12. *Yeasts*															
Yeast, fresh	Compressed	0	4·0	0·1	2·3	27	7	Trace	—	0	23	0·50	7·3	0	0
Brewer's yeast	Dried	0	11·1	0·3	13·7	100	(50)	(5·7)	—	0	300–1,500	1·37	11–22	0	0
Food yeast (*torula utilis*)	Dried	0	12·2	0·7	0·9	59	36	5·7	—	0	188	1·42	11·6	0	0
13. *Cakes and Buns*															
Bun	Plain or currant	0	2·3	2·2	13·9	85	10	0·6	75	0	14	(0·03)	(0·3)	0	0
Cake, currant		0	2·3	5·1	13·6	110	17	0·5	70	77	10	(0·01)	(0·1)	0	0
Cake, plain		0	2·8	1·4	16·2	89	14	0·6	50	85	11	(0·01)	(0·1)	0	0
Doughnuts		0	2·3	4·3	11·9	96	14	0·5	40	0	8	(0·01)	(0·1)	0	0
Jam tart	Short pastry	0	1·0	3·8	15·6	101	4	0·3	85	0	(10)	(0·01)	(0·1)	0	0
Scone		0	2·3	2·8	11·4	80	33	0·4	60	0	15	(0·02)	(0·3)	0	0
14. *Meat, Fish and Egg Dishes*															
Beefsteak pudding		0	2·9	3·7	5·5	67	2	0·5	540	0	(3)	(0·04)	(0·7)	0	0
Fish cakes		0	3·5	4·0	2·5	60	6	0·2	300	0	(3)	(0·02)	(0·4)	0	0
Sausage roll, or meat pie	Short pastry	0	3·1	6·5	8·0	103	4	0·6	300	0	9	(0·03)	(0·5)	0	0
Scrambled egg or omelette		0	2·9	7·1	0·2	79	17	0·6	800	(230)	(7)	(0·07)	0	0	(14)
15. *Puddings*															
Baked sponge pudding		0	1·8	6·8	13·7	123	6	0·2	60	0	(17)	(0·01)	0·1	0	0
Milk pudding		0	1·1	1·6	5·3	40	35	Trace	40	(23)	(3)	(0·01)	Trace	0	0
Steamed pudding	With raisins	0	1·1	4·4	0·6	86	15	0·2	60	0	(8)	(0·01)	0·1	0	0
Custard	Pouring	0	0·9	1·1	4·5	31	35	0	50	(30)	(3)	(0·03)	0	0	0
Custard	Egg, baked	0	1·5	1·7	2·5	31	36	0·1	50	(60)	(4)	(0·04)	0	0	2
16. *Sauces and Soups*															
Sauce, white	Savoury	0	1·1	2·8	2·5	40	32	Trace	380	(30)	(3)	(0·03)	Trace	0	0
Soup, mixed	As served	0	0·6	0·4	1·1	10	10	0·4	170	(70)(c)	(1)	0	0	(2)	0
Dried soup, meat and vegetable	Powder	0	7·9	4·8	13·0	125	30	2·2	1,400	4,800(c)	90	(0·03)	1·1	12	0

(c) The sole source of vitamin A potency is carotene.

NOTES ON THE TABLE OF NUTRITIVE VALUE OF FOODSTUFFS

The uses and limitations of Tables of food values have been discussed in Chapter XXI. The following notes on the Table of Nutritive Value of Foodstuffs on pp. 427 to 435 serve to explain certain matters of detail relating to the Table and to assist the reader in its use for the different types of dietary analysis it is designed to facilitate.

1. General Notes

(*a*) The values given for the different nutrients are those presen in one ounce of the edible portion of foodstuffs either raw o cooked.

(*i*) *Raw Foodstuffs.* The nutritive value of raw foodstuffs ha been included to enable analyses to be made from dietar surveys in institutions, etc., where the weights of the raw foodstuffs entering the institution or household have to b used in the calculations. In assessing the nutritive valu of such diets, allowance must be made for inedible wast as described in note 2 below.

(*ii*) *Cooked Foodstuffs.* In drawing up diets for individua patients, the amounts of each foodstuff to be eaten by th patient are prescribed, and the weight or size of eac serving will, of course, be that of the cooked foodstuff t be presented to the patient. For individual dietaries, therefore, it is the values given for cooked foodstuffs whic must be used in assessing the nutritive value of the die as a whole.

(*b*) In practically every instance, a single value is given for eac nutrient instead of a range of values. It is well known tha the chemical composition of many foodstuffs may show wid variations depending on the variety, environment and seaso in which the food was produced, the stage of growth, and s forth. The values given are the average of a range of samples but it must be emphasized that they are average values an that as a result they may vary somewhat from those obtaine by analysis of a foodstuff in any particular area or season Therefore, dietary analyses based on the data presented i the Table provide only an approximation to the actual composition of the diet. At the same time, it is considered tha the insertion of only a single value for each nutrient rende the Table more generally useful than the inclusion of a rang of values and that the approximation to the actual composition of the diet obtained by the use of the table sufficiently accurate for the purposes of practical dietetics.

(*c*) In the Table a dash indicates that the value for any nutrient approximate percentage waste is unknown. Where the valu has been calculated from the known value for a closel related foodstuff, the figure is enclosed in brackets.

The vitamin contents of the cooked foodstuffs in the Tab have been obtained either from analyses of cooked foo given in the literature or by the application of factors to t values given for the corresponding raw foodstuffs, the factors allowing for the loss of vitamins and changes moisture content brought about in foodstuffs by cooking processes.

2. ALLOWANCE FOR INEDIBLE WASTE

Many foodstuffs as purchased contain inedible portions, such as the bones of meat, stalks of vegetables, etc., and allowance must be made for this when using a Table in which the data refer to the edible portion of the foodstuffs.

To obtain the nutritive value of a foodstuff containing inedible waste, an appropriate deduction for this waste must therefore be made from the 'as purchased' weight before the nutritive value of the foodstuff is calculated from the Table.

To enable this to be done, a column headed 'Percentage of inedible waste' has been included in the Table.

The figures in this column represent the average amount of the inedible waste in the foodstuffs as purchased.

Example. – The weight of meat in a survey in which raw foodstuffs are being used to assess the nutritive value of a diet is 10 oz. per head. In the column 'Percentage of inedible waste', the approximate amount of waste in good quality meat is shown as 17 per cent. Therefore the 10 oz. of meat represent only 8·3 oz. of edible material and the nutritive value of the meat is therefore obtained by multiplying the figures for the amount of nutrients in 1 oz. of meat by 8·3 and not by 10.

The only exceptions to this procedure in the attached Table are the composite values for 'average' fresh and canned vegetables and 'average', fresh, canned, and dried fruit. For these items, the nutritive value represents that obtainable from the 'as purchased' weight, appropriate allowances having been made for the inedible waste in the individual vegetables and fruit when arriving at the averages.

3. COMPOSITE FIGURES FOR VEGETABLES AND FRUIT

In dietary surveys over a period it may be difficult to ascertain the various vegetables and fruit which have entered into the diet, and it will be tedious to calculate the nutritive value of each, even if known. In both large-scale surveys and individual dietary analyses it is justifiable to use a composite figure for vegetables and fruit, and for this reason average values are given in the Table for raw and boiled fresh vegetables, canned vegetables, raw and stewed fresh fruit, canned fruit, and dried fruit. As indicated, in paragraph 2 above, allowance has been made for inedible waste in these composite figures.

The average values have been calculated from the nutritive value of the vegetables and fruit normally obtainable in Great Britain in peacetime. For countries where the predominating varieties of vegetables and fruit differ widely from those obtainable in Great Britain, composite values will have to be calculated from the figures given in the Table for the individual vegetables and fruit.

4. LOSSES IN COOKING AND AT TABLE

Losses in the nutritive value of the edible portion of a diet occur both in the inevitable wastage of foodstuffs in cooking and at table and in the destruction of certain of the nutrients brought about by cooking processes.

(*a*) *Loss of vitamins in cooking.* The most marked reduction in the nutritive value of a diet through cooking occurs among the vitamins. The extent of the loss varies with the cooking method employed, the time of cooking, the volume of cooking water in relation to the foodstuff, etc. The following arbitrary figures represent the average percentage loss of vitamins in an average mixed diet.

Vitamin A, 5 per cent.	Nicotinic Acid, up to 20 per cent.
Vitamin B_1, 15 per cent.	Ascorbic Acid, 60 per cent.
Riboflavin, 10 per cent.	

(*b*) *Loss of nutrients other than vitamins.* Here the loss is chiefly due to the cooking and plate wastage of food and is much smaller than in the case of the vitamins. It will depend on factors such as cooking methods, feeding habits, etc., and is impossible to estimate empirically with any degree of accuracy. Such loss tends to be higher in institutions, etc., where large numbers of individuals are being fed together, and losses varying from 1 to 10 per cent have been recorded from different institutions. An arbitrary figure of 10 per cent is often adopted.

The dietary standards which have been laid down at various times by authoritative bodies refer to the amount of nutrients which must actually be ingested by the individual. Therefore, before the nutritive value of a diet which has been calculated from the raw foodstuffs composing it can be compared with dietary standards, allowance must be made for cooking and plate wastage by making the appropriate deductions indicated in sub-paragraphs (*a*) and (*b*) above from the amounts of the different nutrients present in the raw diet.

5. LOSSES FROM NON-ABSORPTION OF FOODSTUFFS IN THE ALIMENTARY TRACT

The food finally entering the alimentary tract is never completely digested and absorbed there. For instance, indigestible fibres in animal and plant food material interfere with the absorption of digested food material. The loss of food through non-absorption varies according to the type of diet, but it has been calculated that in an average mixed diet, protein, fat, and carbohydrate have a nett energy value of 4, 9, and 4 Calories per gram respectively. In the Table, allowance has been made for non-utilization of ingested foodstuffs by using the physiological factors mentioned above for the Caloric value of protein, fat, and carbohydrate, instead of the factors 4·1, 9·3 and 4·1, which are correct only if complete absorption of ingested foodstuff occurs.

6. VITAMIN A VALUES

The vitamin A potency of plant foodstuffs is due to the presence of carotene, which in man is less well absorbed than pre-formed vitamin A from animal sources. There also appears to be considerable variation in the extent to which carotene is absorbed from the different kinds o plant foodstuffs. The International Standard of 0·6 microgrammes o beta-carotene = 1 International Unit of vitamin A potency assume complete absorption of carotene, and therefore does not represent the vitamin A potency of plant tissues in human diets. It has, however, been agreed provisionally that in man, over the range of plant foodstuff normally consumed, only about one-third of the carotene in these foodstuffs is absorbed and utilized. Therefore in order to express the carotene content of a diet in terms of vitamin A potency, it is necessary to divide by three the sum of the International Units theoretically obtainable from the carotene. It is believed that the vitamin A potency of plan tissues calculated on this basis is reasonably comparable with the potency of vitamin A from animal sources, so that if the conversion factor mentioned above is used, the vitamin A potency of plant and animal foodstuffs can be added together and expressed as one total. To facilitate the making of this correction, all the vitamin A values in the table that are derived solely from carotene are marked with the letter (c), and it is the sum of the vitamin A values so marked that must be divided by three when calculating the vitamin A potency of a mixed diet.

7. References

The Table has been compiled from the following sources of information:

(*a*) *Standard works*
  (i) *Nutritive Value of Wartime Foods*, Medical Research Council, Memorandum No. 14, H.M. Stationery Office, London 1945.
  (ii) *Chemical Composition of Foods*, McCance and Widdowson, Medical Research Council, H.M. Stationery Office, London, 1940.
  (iii) *Food and Beverage Analysis*, Bridges and Mattice, Kimpton, London, 1942.
  (iv) *The Avitaminoses*, Eddy and Dalldorf, Baillière, Tindall, and Cox, London, 1941.

(*b*) *Current literature.*
  (i) *The Nutritive Value of Indian Foods*, Health Bulletin No. 23, Government of India, 1941.
  (ii) *Food Composition Tables, Bulletin No. 178*, Council for Scientific and Industrial Research, Commonwealth of Australia, 1944.
  (iii) *Table of the Vitamin Content of Human and Animal Foods*, Fixsen and Roscoe, Nutrition Abstracts and Reviews, **9**, 795–861, 1939–40.
  (iv) *The Vitamin Content of Human Foods as affected by Processes of Cooking and Canning*, Fixsen, Nutrition Abstracts and Reviews, **8**, 281–307, 1938–39.
  (v) *Distribution of Nicotinic Acid in Human and Animal Foods*, Bacharach, Nutrition Abstracts and Reviews, **10**, 459–465, 1940–41.
  (vi) *Nicotinic Acid Content of Foodstuffs*, McVicar and Berryman, Journal of Nutrition, **24**, 235–243, 1942.
  (vii) *Studies of the Average American Diet. – Riboflavin, Nicotinic Acid, and Pantothenic Acid Content*, Cheldelin and Williams, Journal of Nutrition, **26**, 417, 1943.
  (viii) Numerous other references in the recent literature to the analysis of individual foodstuffs.

TABLE 18

VEGETABLES – FOOD VALUES

1 to 3 per cent CARBOHYDRATE. Average value –
1 oz. = 0·6 grams CHO.

Beans, French (boiled).
Beans, Runner (boiled and canned).
Brussels sprouts (boiled and canned).
Cabbage (raw and boiled).
Carrot (raw and boiled).
Cauliflower (boiled).
Celery (boiled).
Cucumber (raw).
Egg plant.
Endive (raw).
Leeks (boiled).
Onion bulb (boiled).
Radish (raw).
Spinach (boiled and canned).
Swedes (boiled).
Tomato (raw and fried).
Turnip (boiled).
Vegetable Marrow (boiled).
Watercress (raw).

100 grams (3½ oz.) contain approximately Prot. 0·3 grams,
CHO 2·2 = Cals. 10.

6 per cent CARBOHYDRATE. 1 oz. = 2 grams CHO.

Beans, broad (boiled).
Kale (boiled).
Onion, spring (raw).
Peas, green, fresh (boiled and canned).
Pepper, red, Chillies, Pimento, fresh.

100 grams (3½ oz.) contain approximately Prot. 1 gram,
CHO 7 = Cals. 32.

10 per cent CARBOHYDRATE. 1 oz. = 3 grams CHO.

Beetroot (boiled).
Parsnips (boiled).

100 grams (3½ oz.) contain approximately Prot. 0·4 grams,
CHO 10·5 = Cals. 44.

12 to 15 per cent CARBOHYDRATE. Average value –
1 oz. = 4·2 grams CHO.

Beans, baked (canned).
Beans, haricot (boiled).
Lentils (boiled).
Peas, dried (boiled).
Potatoes, fresh (boiled).

100 grams (3½ oz.) contain approximately Prot. 1·7 grams,
CHO 15 = Cals. 66.

When fresh vegetables are out of season, tinned ones may be substituted.

TABLE 19

FRUIT: CARBOHYDRATE CONTENT – RAW AND COOKED

1 to 3 per cent CARBOHYDRATE. Average value –
1 oz. = 0·6 grams CHO.

Apple, stewed without sugar.
Avocado Pear, raw.
Cherry, stewed without sugar.
Currants, black, stewed.
Currants, red, raw, and stewed without sugar.
Gooseberries, stewed without sugar.
Grapefruit, raw.

Lemon.
Loganberries, raw.
Melon, yellow.
Plums + stones, stewed without sugar.
Raspberries, raw, and stewed without sugar.
Rhubarb, stewed without sugar.

100 grams (3½ oz.) contain approximately Prot. 0·6.
CHO 2·2 = Cals. 11.

5 per cent Carbohydrate. 1 oz. = 1·5 grams CHO.

Apple, baked.
Apricot, fresh.
Blackberries, raw.
Currants, black, raw.
Damsons, stewed without sugar.
Orange, sweet.
Pear, cooking, stewed without sugar.
Plum, dessert, raw.

100 grams (3½ oz.) contain approximately Prot. 0·6,
CHO 5·2 = Cals. 23.

7½ to 10 per cent Carbohydrate. Average value – 1 oz. = 2·5 grams CHO.

Apple, English, eating.
Apricot, canned.
Banana, raw.
Cherries, raw.
Cape gooseberries, raw.
Damsons, raw.
Gooseberries, eating, raw.
Greengages, raw, and + stones, stewed without sugar.
Lime.
Mango, ripe.
Orange juice.
Peach, fresh, raw.
Pear, eating, raw.

100 grams (3½ oz.) contain approximately Prot. 0·6,
CHO 9·1 = Cals. 39.

12 to 14 per cent Carbohydrate. Average value – 1 oz. = 4 grams CHO.

Grapes, green, raw.
Grapefruit, canned.
Peach, canned.
Pear, canned.
Plantain.
Prunes + stones, stewed without sugar.

100 grams (3½ oz.) contain approximately Prot. 0·2,
CHO 14 = Cals. 57.

15 per cent Carbohydrate. 1 oz. = 4·5 grams CHO.

Apricot, dried, stewed without sugar.
Peach, dried, stewed without sugar.
Pineapple, canned.
Plum, canned.

100 grams (3½ oz.) contain approximately Prot. 0·9,
CHO 15·7 = Cals. 67.

25 per cent Carbohydrate. 1 oz. = 7·5 grams CHO.

Figs, dried, stewed without sugar.

100 grammes (3½ oz.) contain approximately Prot. 2·1,
CHO 26 = Cals. 112.

N.B. Canned fruit, owing to the high sugar content of the syrup, must not be used in any diet where the carbohydrate intake is being prescribed quantitatively.

TABLE 20

CONVERSION TABLE FOR FRUITS – 10-GRAM CARBOHYDRATE PORTIONS

5 per cent Fruit 7 oz.	7½ per cent – 10 per cent Fruit 4 oz.		12 per cent Fruit 3 oz.	15 per cent Fruit 2½ oz.
Apple, baked Apricot, fresh Blackberries, raw Currants, black, raw Damsons, stewed without sugar Oranges, sweet Pear, cooking, stewed without sugar Plum, dessert, raw	Apple, English eating Damsons, raw Gooseberries, eating, raw Greengages + stones, stewed without sugar Mango, ripe Orange juice Peach, fresh, raw Pear, eating, raw	Apricot, canned Banana, raw Cherries, raw Cape gooseberries, raw Greengages, raw	Grapes, green, raw Grapefruit, canned Peach, canned Pear, canned Plantain Prunes + stones, stewed without sugar	Apricot, dried, stewed without sugar Peach, dried, stewed without sugar Pineapple, canned Plum, canned

Note. – Conversion factors are not given for fruits containing 1 per cent to 3 per cent of carbohydrate (see Table 19), since the servings required to provide 10 grams of carbohydrate would be excessively large. A satisfactory conversion factor for these fruits will be provided by a reasonable serving of any of them to which a supplement of ½ oz. sugar is added.

TABLE 21

APPROXIMATE 100-CALORIE PORTIONS

1. CARBOHYDRATE. All contain 20 to 25 grams carbohydrate.
   1½ oz. (45 grams) – Bread, 1 medium slice.
   1¾ oz. (52 grams) – Dates, 5 large – E.P.*
   3½ oz. (105 grams) – Macaroni or Spaghetti, boiled, ½ breakfast cup.
   5 oz. (150 grams) – Potato, fresh boiled, 2 medium.
   6 oz. (180 grams) – Prunes, stewed, 10 large – A.P.*
   7 oz. (210 grams) – Banana, 2 large – E.P.
   10 oz. (300 grams) – Orange juice, ½ pint.
   20 oz. (600 grams) – Carrot, boiled, 3 large servings.

2. PROTEIN. All contain approximately 15 to 16 grams protein.
   1¾ oz. (52 grams) – Lean meat, average roast and boiled.
   2 oz. (60 grams) – Fowl, average roast and boiled.
   2¼ oz. (67 grams) – Kidney, stewed.
   2¼ oz. (67 grams) – Cheese, Cheddar, less ¾ oz. butter.
   2½ oz. (75 grams) – Duck, roast, less ½ oz. butter.
   2½ oz. (75 grams) – Sweetbread, stewed, less ⅛ oz. butter.
   2½ oz. (75 grams) – Herring, fried in oatmeal, less ¼ oz. butter.
   2¾ oz. (82 grams) – Cod roe, fried in crumbs, less ½ oz. butter.
   2¾ oz. (82 grams) – White fish, steamed + ⅛ oz. butter or dessertspoonful of thin white sauce.
   2¾ oz. (82 grams) – Shellfish, crab or lobster, boiled + 1 dessertspoonful of mayonnaise.
   3¼ oz. (97 grams) – Herring, baked in vinegar, less ¼ oz. butter.

3. FAT. All contain approximately 12 grams fat.
   ½ oz. (15 grams) – Butter or vitaminized margarine.
   ¾ oz. (22 grams) – Bacon, fried or grilled.
   1 oz. (30 grams) – Cream, 40 per cent.
   2 oz. (60 grams) – Cream, 20 per cent.

4. FOODS CONTAINING CARBOHYDRATES, PROTEIN AND FAT IN VARYING AMOUNTS.
   ⅞ oz. (26 grams) – 2 level tablespoons oatmeal.
   ⅞ oz. (26 grams) – Cheese, Cheddar.
   1¼ oz. (37 grams) – Pulses, peas, beans, lentils, dried.
   4½ oz. (135 grams) – Fresh peas and beans, cooked.
   6 oz. (180 grams) – Whole milk.
   11 oz. (330 grams) – Skimmed milk.

* E.P. denotes 'Edible Portion': A.P. 'As Purchased'.

Since the ordinary housewife will find it impracticable to weigh such small quantities of butter as ⅛ and ¼ oz., it is suggested that she divides 1 oz. of butter into eight equal pats, and that she adds or deducts one or more of such pats as required. ⅛ an ounce of butter in the form of a round pat has approximately the diameter of a sixpence.

TABLE 22

List of Common Foods, 1 oz. Portions in Descending Order of Caloric Value

	Prot. grams	Fat grams	CHO grams	Calories
30 *grams* (1 *oz.*) *contain approximately:*				
Olive oil	—	28·4	—	256
Butter or vitaminized margarine	0·1	23·4	—	211
Bacon, raw, home cured – A.P.	2·7	11·2	—	112
40 per cent cream	0·5	11·9	0·6	111
Oatmeal (dry weight)	3·4	2·5	18·6	111
Benger's Food	3	0·3	24	111
Cheddar cheese – A.P.	6·7	9·3	—	110
Sugar, glucose	—	—	27	108
Oatcake	3·7	3	15	102
Cornflour	0·2	0·1	24·4	99
Rice, polished	1·8	0·3	22·2	99
Wheat, shredded	3·9	0·7	18·7	97
Egg, one whole	6·2	5·8	0·5	79
Bread, white	2·3	0·2	15·6	73
Jam	0·1	—	17·6	71
Bread, wholemeal	3·1	0·6	11·2	65
Lean meat, cooked	8·5	3·1	—	63
20 per cent cream	1	6	1	62
Chicken, average roast and boiled	7·9	2·5	—	54
Shellfish, crab or lobster, boiled	5·7	1·1	—	33
25 per cent fruit, *e.g.* figs, dried, stewed	0·6	—	7·6	33
White fish, steamed	5·5	0·5	—	26
15 per cent fruit, *e.g.* apricot, dried, stewed	0·6	—	4·6	21
Potatoes, fresh, boiled	0·4	—	4·9	21
Milk	0·9	1	1·2	17
12 per cent fruit, *e.g.* prunes, stewed	0·3	—	4	17
10 per cent fruit, *e.g.* banana, raw	0·2	—	2·9	12
7½ per cent fruit, *e.g.* apple, eating	0·08	—	2·4	10
6 per cent vegetable, *e.g.* onion, spring, raw	0·2	—	1·7	8
3 per cent vegetable, *e.g.* carrot, boiled	0·2	—	1·1	5
3 per cent fruit, *e.g.* apple, stewed	—	—	1·1	4

TABLE 23

AVERAGE CALORIC VALUES AND ALCOHOL CONTENTS OF VARIOUS BEVERAGES

	Vol.	Per cent Alcohol by weight	Total Sugar Content in grams	Total Calories
Brandy, cordial glass	⅔ oz.	35	0	47
Whisky, large glass	2 oz.	35	0	140
Rum, small glass	1 oz.	35	0	70
Gin, small glass	1 oz.	35	0	70
Cocktail, large, 50 per cent gin 50 per cent vermouth	2 oz.	30	3	140
Liquers, cordial glass	⅔ oz.	30	5·5	65
Fortified wines (sherry, port, etc.), small glass	1 oz.	15	1·3	35
Light wines, claret glass	4 oz.	10	1·2	80
Cider, bottled	½ pint	5	3·5	115
Ale, strong	½ pint	6	10	160
Beer	½ pint	3·5	5	100
Stout	½ pint	4	10	130

1 c.c. ethyl alcohol = 7 Calories.

Note: Details of the nutritive value of these beverages will be found in the Table of Nutritive Value of Foodstuffs on p. 435.

TABLE 24

LIST OF COMMON FOODS AS HELPINGS AND HANDY MEASURES

MEAT AND FISH
1½ to 2 oz. = small helping.
3 oz. = average ,,
4 to 5 oz. = large ,,
Butter, ¾ oz. = 1 level tablespoonful.
Dripping, ¾ oz. = 1 ,, ,,
Olive oil, ½ oz. = 1 tablespoonful.

BREAD 1 oz. = 1 thin slice 5″ × 4½″ × ¼″.

VEGETABLES
Cabbage or other green vegetables 5 to 7 oz. = large helping.
Carrot and turnip 3½ oz. = average helping.
Boiled potato 3½ oz. = 1 medium or 2 small.

SUGAR 3 oz. = 1 tablespoonful.
5 grams = 1 average lump.

MILK PUDDING from cereal ½ oz.
Milk 5 oz. = average helping.
¾ oz. oatmeal makes 3 tablespoonfuls porridge.

TABLE 25

EXCHANGES IN DIETS FOR DIABETES

1. (*a*) FOR 1 EGG:
    - Average cheese = 1 oz. less ⅛ oz. butter from ration.
    - Sardines, oil drained away = 1 oz.
    - Gammon (ham) boiled, salted = 1¾ oz. less ¼ oz. butter from ration.
    - Herring, fried in oatmeal = 1 oz.
    - ,, baked in vinegar = 1¼ oz.
    - Liver, fried = ¾ oz.
    - Kidney, stewed = ¾ oz. add ¼ oz. butter to ration.
    - Fowl, average roast and boiled = ¾ oz. add ⅛ oz. ,, ,,
    - White fish, steamed = 1¼ oz. add ¼ oz. ,, ,,
    - Beef, average roast and boiled = ¾ oz. add ⅛ oz. ,, ,,
    - Bacon, raw = 2¼ oz. less ¾ oz. butter from ration.

    (*b*) FOR 1 EGG + 1¼ OZ. BACON:
    - Gammon (ham) boiled, salted = 2½ oz.
    - Herring, fried in oatmeal = 1½ oz. add ½ oz. butter to ration.
    - ,, kippered = 3 oz. add ½ oz. ,, ,,
    - Kidney, stewed = 1¼ oz. add ¾ oz. ,, ,,
    - White fish, steamed = 1¾ oz. add ¾ oz. ,, ,,
    - Finnan haddock = 3½ oz. add ¾ oz. ,, ,,
    - Bacon, raw = 2¼ oz.
    - ,, fried or grilled = 1¾ oz.
    - Tongue (canned) = 1¾ oz. add ½ oz. butter to ration.
    - Pilchards, canned in tomato sauce = 2 oz. add ½ oz. ,, ,,

2. FOR LEAN MEAT, 3 OZ.:
    - Herring, fried in oatmeal = 4 oz. less ¼ oz. butter from ration
    - Salmon (canned) = 4½ oz. less ⅛ oz. ,, ,,
    - Tripe, stewed = 5 oz. add ¼ oz. butter to ration.
    - Chicken, average roast and boiled = 3¼ oz.
    - Rabbit, stewed = 4 oz.
    - Liver, fried = 3 oz. less ⅛ oz. butter from ration.
    - Cod roe, fried in crumbs = 4¼ oz. less ¼ oz. ,, ,,
    - Sweetbread, stewed = 4 oz.
    - White fish, steamed = 4¾ oz. add ¼ oz. butter to ration.
    - Duck, roast = 4 oz. less ¾ oz. butter from ration
    - Corned beef = 3½ oz. less ¼ oz. ,, ,,
    - Roast pork = 4 oz. less 1⅛ oz. ,, ,,
    - Tongue (canned) = 4¾ oz. less ¾ oz. ,, ,,

3. FOR WHITE FISH, 8 OZ.:
    - Salmon (canned) = 3 oz. less ¼ oz. butter from ration
    - Chicken, roast = 2 oz. less ⅛ oz. ,, ,,
    - ,, boiled = 2¼ oz. less ¼ oz. ,, ,,
    - Rabbit, stewed = 2½ oz. less ⅛ oz. ,, ,,
    - Sweetbread, stewed = 2½ oz. less ¼ oz. ,, ,,
    - Herring, baked in vinegar = 8½ oz. less ½ oz. ,, ,,

Meat, lean only, average roast and boiled = 2 oz. less ¼ oz. butter from ration.
Tripe, stewed = 3¼ oz.
Gammon (ham) boiled, salted, lean only = 3½ oz. less ¼ oz. ,, ,,
Tongue (canned) = 3 oz. less ¾ oz. ,, ,,
Eggs = 2½ oz. less ½ oz. ,, ,,

4. For 1 oz. National Bread = 1 thin slice = approximately 15 grams carbohydrate.

Where weight is given, exchanges *must* be weighed accurately.

Macaroni (boiled) = 2½ oz.
Shredded wheatflakes = ¾ oz.
Biscuits: 3 cream crackers.
2 rich tea biscuits.
3 water biscuits.
2 pieces Ryvita.
Prunes, stewed without sugar = 3¾ oz.
Apple, eating = 6 oz.
Banana, raw = 5 oz.
Potato, cooked = 3 oz.
Rice (dry weight) = ¾ oz.
Benger's Food (dry weight) = ¾ oz.
Oatmeal = ¾ oz.

The exchanges are unaffected if vitaminized margarine is used as an alternative for butter. See footnote, Table 21, for fractions employed.

DIET SHEETS

DIET No. 1

Diabetes

Sub-basal (approximately 1,200 Calories) diet for initial treatmen of mild cases not requiring Insulin. This diet should not be used fc more than 7 to 10 days.

Prot. 50	Fat 84	CHO 65	= Calories 1,214
Total 'G'	= 102	'G : F.A.' :: 1 : 0·97	
,, 'F.A.'	= 99		

	Prot.	Fat	CHO	Calori
Breakfast				
Bacon, 2¼ oz, or Exchange No. 1 (*b*)	6·1	25·4	—	25
Tomato or other low carbohydrate vegetable, 3½ oz. . . .	0·9	—	2·1	1
National bread, 1 oz. or equivalent.	2·4	0·4	14·6	7
Tea or coffee. Milk from ration.				
	9·4	25·8	16·7	3
Dinner				
Clear soup, Marmite or Bovril .	0·3	—	—	
Lean meat, 2 oz., or Exchange No. 3	17	6·2	—	1
Cabbage or other low carbohydrate vegetable, 7 oz. . . .	1·4	—	2·1	[illegible]
Curds with milk from ration.				
	18·7	6·2	2·1	1
Tea				
1 egg or Exchange No. 1 (*a*) . .	6·2	5·8	0·5	[illegible]
Tomato, lettuce, cress, etc., as salad, 3½ oz.	0·9	—	2·1	[illegible]
National bread, 1 oz. or equivalent	2·4	0·4	14·6	[illegible]
Tea with milk from ration.				
	9·5	6·2	17·2	1
Supper				
National bread, 1 oz. or equivalent	2·4	0·4	14·6	[illegible]
Tomato or other low carbohydrate vegetable, 3½ oz. . . .	0·9	—	2·1	1
Milk from ration.				
	3·3	0·4	16·7	
Bed-time				
Cup of Marmite	0·3	—	—	
Daily milk ration, 10 oz. . . .	9	10	12	1
Daily butter (or vitaminized margarine) ration, 1½ oz.	0·1	35·1	—	8
Total . .	50·3	83·7	64·7	1,2

DIET No. 1A. – More Expensive

Diabetes

Sub-basal (approximately 1,200 Calories) diet for initial treatment of mild cases not requiring Insulin. This diet should not be used for more than 7 to 10 days.

Prot. 52 Fat 82 CHO 65 = Calories 1,206
Total 'G' = 103 'G : F.A.' :: 1 : 0·95
,, 'F.A.' = 98

	Prot.	Fat	CHO	Calories
BREAKFAST				
Tomato or other low carbohydrate vegetable, 3½ oz.	0·9	—	2·1	12
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea or coffee.				
Milk and cream from ration.				
	3·3	0·4	16·7	84
FORENOON				
Cup of Marmite	0·3	—	—	1
1 water biscuit	0·8	0·2	5·3	26
	1·1	0·2	5·3	27
LUNCH				
Fillet of sole or other white fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Melted butter from ration with ¼ oz. chopped parsley	0·3	—	—	1
Salad of tomato, lettuce, cress, 4 oz. or other low carbohydrate vegetable	1	—	2·4	14
Curds with milk from ration.				
Serve with whipped cream.				
	17·8	1·5	2·4	94
TEA				
2 water biscuits or equivalent	1·6	0·4	10·6	52
Tomato, lettuce, cress, 3½ oz.	0·9	—	2·1	12
Tea with milk and cream from ration.				
	2·5	0·4	12·7	64
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	1
Chicken, roast, 2 oz., or Exchange No. 3	16·8	4·2	—	105
Salad or other low carbohydrate vegetable, 3½ oz.	0·9	—	2·1	12
Potato, boiled, 2 oz.	0·8	—	9·8	42
Fruit as stewed apple, 4 oz., or orange, 2¾ oz.	—	—	4·4	18
Coffee – milk and cream from ration.				
	18·8	4·2	16·3	178
Daily milk ration, 10 oz.	9	10	12	174
Daily butter (or vitaminized margarine), 2 oz.	0·2	46·8	—	422
Daily 40 per cent cream, 1½ oz.	0·7	17·8	—	163
TOTAL	53·4	81·3	65·4	1,206

DIET No. 2

Diabetes

Basal (approximately 1,500 Calories) diet with approximately 80 grams Carbohydrate for mild cases not requiring Insulin.

	Prot.	Fat	CHO	Calories
To Diet No. 1 or No. 1A (more expensive) *add*	50·3	83·7	64·7	1,214
National bread, 1 oz.	2·4	0·4	14·6	72
Butter (or vitaminized margarine), 1 oz.	0·1	23·4	—	211
TOTAL	52·8	107·5	79·3	1,497

or,

		Prot.	Fat	CHO	Calories
Milk Pudding	Cereal, ⅓ oz.	—	—	8·1	32
	Milk, 5½ oz.	4·9	5·5	6·6	95
	Saccharin				
	Butter (or vitaminized margarine), ¾ oz.	0·1	17·5	—	158
Diet No. 1		50·3	83·7	64·7	1,214
	TOTAL	55·3	106·7	79·4	1,499

or,

	Prot.	Fat	CHO	Calories
15 per cent fruit as dried apricot, stewed, 2 oz.	1·2	—	9·2	42
1 egg white	3·1	—	—	12
1 water biscuit	0·8	0·2	5·3	26
Butter (or vitaminized margarine), 1 oz.	0·1	23·4	—	211
Diet No. 1A	53·4	81·3	65·4	1,206
TOTAL	58·6	104·9	79·9	1,497

DIET No. 3

Diabetes

Maintenance diet. Approximately 1,850 Calories and 95 grams Carbohydrate, for mild cases suitable for treatment without Insulin.

	Prot.	Fat	CHO	Calories
BREAKFAST				
Porridge from ¾ oz. oatmeal or equivalent	2·5	1·8	13·9	82
Bacon, 1¼ oz. and 1 egg, or Exchange No. 1 (*b*)	9·6	19·9	0·5	219
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk from ration.				
	14·5	22·1	29	373
DINNER				
Clear soup, Marmite or Bovril .	0·3	—	—	1
Lean meat, 3 oz., or Exchange No. 2	25·5	9·3	—	186
Cabbage or other low carbohydrate vegetable, 7 oz. . . .	1·4	—	2·1	14
Milk pudding from ½ oz. cereal (milk from ration)	0·1	—	12·2	49
or other 12 grams carbohydrate sweet (see pp. 423 to 424)				
	27·3	9·3	14·3	250
TEA				
White fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Melted butter from ration as sauce with ¼ oz. chopped parsley. .	0·3	—	—	1
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Salad or other low carbohydrate vegetable, 5 oz. . . .	1	—	3	16
Tea with milk from ration.				
	20·2	1·9	17·6	168
SUPPER				
3 water biscuits or equivalent .	2·4	0·6	15·9	79
Tomato, 1 oz.	0·3	—	0·6	4
Milk from ration.				
	2·7	0·6	16·5	83
BED-TIME				
1 cup Marmite	0·3	—	—	1
Daily milk ration, 15 oz. . . .	13·5	15	18	261
Daily butter (or vitaminized margarine) ration, 3⅓ oz.	0·3	78	—	703
TOTAL . .	78·8	126·9	95·4	1,839

DIET No. 3A. – More Expensive

Diabetes

Maintenance diet. Approximately 1,850 Calories and 95 grams Carbohydrate, for mild cases suitable for treatment without Insulin.

	Prot.	Fat	CHO	Calories
BREAKFAST				
½ grapefruit	0·3	—	2·4	11
2 eggs or Exchange No. 1(*a*)	12·4	11·6	1	158
National bread, 1½ oz. or equivalent	3·6	0·6	21·9	107
Tea or coffee.				
Milk and cream from ration.				
	16·3	12·2	25·3	276
LUNCH				
Chicken, roast, 2 oz., or Exchange No. 3	16·8	4·2	—	105
Salad or other low CHO vegetable, 7 oz.	1·4	—	4·2	22
Fruit as prunes, stewed, 4 oz.	1·2	—	16	69
Whipped cream from ration.				
	19·4	4·2	20·2	196
TEA				
2 water biscuits or equivalent	1·6	0·4	10·6	52
Butter from ration.				
⅛ oz. Marmite	0·3	—	—	1
Tea with cream from ration.				
	1·9	0·4	10·6	53
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	1
Lean meat, 3 oz., or Exchange No. 2	25·5	9·3	—	186
Salad or other low CHO vegetable, 4 oz.	1	—	2·4	14
Fruit as dried apricot, stewed, 4 oz.	2·4	—	18·4	83
1 water biscuit	0·8	0·2	5·3	26
Cheddar cheese, ¾ oz.	3·4	4·6	—	55
Coffee with milk and cream from ration.				
	33·4	14·1	26·1	365
Daily milk ration, 10 oz.	9	10	12	174
Daily butter (or vitaminized margarine) ration, 2½ oz.	0·2	58·5	—	527
Daily 40 per cent cream ration, 2¼ oz.	1·2	26·7	1·3	250
TOTAL	81·4	126·1	95·5	1,841

DIET No. 4

Obesity accompanying Mild Diabetes

Approximately 1,500 Calories with 95 grams Carbohydrate.

To reduce caloric value of Diet No. 3 to approximately 1,500 Calories without altering the carbohydrate content, reduce butter or vitaminized margarine from 3⅓ oz. to approximately 2 oz.

	Prot.	Fat	CHO	Calories
Thus: Diet No. 3 . . . . =	78·8	126·9	95·4	1,839
Subtract 1½ oz. butter . . =	0·1	35·1	—	316
TOTAL . .	78·7	91·8	95·4	1,523

To reduce caloric value of Diet No. 3A (more expensive) to 1,500 Calories without altering appreciably the carbohydrate content, omit 1¼ oz. double cream and reduce butter from 2½ oz. to 1½ oz.

	Prot.	Fat	CHO	Calories
Thus: Diet No. 3A (more expensive) . =	81·4	126·1	95·5	1,841
Subtract 1¼ oz. 40 per cent cream =	0·6	14·8	0·7	138
Subtract 1 oz. butter . . =	0·1	23·4	—	211
TOTAL . .	80·7	87·9	94·8	1,492

or reduce butter from 2½ oz. to 1 oz. and omit ¼ oz. double cream.

	Prot.	Fat	CHO	Calories
Thus: Diet No. 3A (more expensive) . =	81·4	126·1	95·5	1,841
Subtract 1½ oz. butter . . =	0·1	35·1	—	317
Subtract ¼ oz. 40 per cent cream =	0·1	2·9	0·1	26
TOTAL . .	81·2	88·1	95·4	1,498

DIET No. 5

Obesity accompanying Mild Diabetes

Approximately 1,200 Calories with 100 grams Carbohydrate.

	Prot.	Fat	CHO	Calories
BREAKFAST				
Porridge from ¾ oz. oatmeal or equivalent	2·5	1·8	13·9	82
1 egg or Exchange No. 2(*a*)	6·2	5·8	0·5	79
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk from ration.				
	11·1	8	29	233
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	1
Chicken, roast, 2 oz., or Exchange No. 3	16·8	4·2	—	105
Cabbage or other low CHO vegetable, 7 oz.	1·4	—	2·1	14
Fruit as prunes, stewed, 3 oz.	0·9	—	12	52
or 12 grams carbohydrate sweet (see pp. 423 to 424)				
	19·4	4·2	14·1	172
TEA				
White fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Chopped parsley, ¼ oz.	0·3	—	—	1
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Salad or other low CHO vegetable, 4 oz.	1	—	2·4	14
Tea with milk from ration.				
	20·2	1·9	17	166
SUPPER				
3 water biscuits or equivalent	2·4	0·6	15·9	79
Milk from ration.				
BED-TIME				
1 cup Marmite	0·3	—	—	1
Daily milk ration, 1 pint	18	20	24	348
Daily butter (or vitaminized margarine) ration, 1 oz.	0·1	23·4	—	211
TOTAL	71·5	58·1	100	1,210

Note. – If the patient can take additional carbohydrate without showing sugar in his urine, 1 or 2 oz. of bread or equivalent may be added to the diet, and ⅓ oz. or ⅔ oz. of butter (or vitaminized margarine) deducted from the butter ration.

DIET No. 5A. – More Expensive

Obesity accompanying Mild Diabetes

Approximately 1,200 Calories with 100 grams Carbohydrate.

	Prot.	Fat	CHO	Calories
BREAKFAST				
Apple, eating, 4½ oz.	0·4	—	10·8	45
1 egg or Exchange No. 1(*a*)	6·2	5·8	0·5	79
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk and cream from ration.				
	9	6·2	25·9	196
LUNCH				
Fillet of sole or other white fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Chopped parsley, ¼ oz	0·3	—	—	1
Salad or other low CHO vegetable, 5 oz.	1·2	—	3	17
Prunes, stewed, 3 oz.	0·9	—	12	52
or 12 grams carbohydrate sweet (see pp. 423 to 424)				
Milk and cream from ration.				
	18·9	1·5	15	149
TEA				
3 water biscuits or equivalent	2·4	0·6	15·9	79
1 oz. tomato } (as sandwich spread)	0·2	—	0·6	3
⅛ oz. Marmite } (as sandwich spread)	0·3	—	—	1
Tea with cream from ration.				
	2·9	0·6	16·5	83
DINNER				
½ grapefruit	0·3	—	2·4	11
Rabbit, stewed, 2½ oz., or Exchange No. 3	16·5	4·8	—	109
Carrot, boiled, 3 oz., or other low CHO vegetable	0·6	—	3·3	16
Garnish, ¼ oz. chopped parsley	0·3	—	—	1
Banana, 4¼ oz., or 12 grams CHO sweet	0·8	—	12·4	53
Coffee.				
Milk and cream from ration.				
	18·5	4·8	18·1	190
Daily milk ration, 1 pint	18	20	24	348
Daily butter (or vitaminized margarine) ration, ½ oz.	—	11·7	—	105
Daily 40 per cent cream, 1¼ oz.	0·6	14·8	0·7	138
TOTAL	67·9	59·6	100·2	1,209

Note. – If the patient can take additional carbohydrate without showing sugar in his urine, 1 or 2 oz. of bread or equivalent may be added to the diet and ⅛ oz. or ⅜ oz. butter (or vitaminized margarine) deducted from the butter ration.

DIET No. 6

Basal Diet for Treatment of Diabetes with Soluble Insulin

Approximately 1,550 Calories with 120 grams Carbohydrate.

	Prot.	Fat	CHO	Calories
INSULIN				
BREAKFAST				
Porridge from ¾ oz. oatmeal or equivalent	2·5	1·8	13·9	82
1 egg or Exchange No. 1(*a*)	6·2	5·8	0·5	79
National bread, 1½ oz.	3·6	0·6	21·9	107
Tea with milk from ration.				
	12·3	8·2	36·3	268
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	1
Rabbit, stewed, 4 oz., or Exchange No. 2	25·2	7·3	—	166
Cabbage or other low carbohydrate vegetable, 7 oz.	1·4	—	2·1	14
Milk pudding {½ oz. cereal milk from ration	0·1	—	12·2	49
or other 12 grams carbohydrate sweet.				
	27	7·3	14·3	230
INSULIN				
HIGH TEA				
White fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Chopped parsley, ¼ oz.	0·3	—	—	1
Potato, boiled, 2 oz. or equivalent	0·8	—	9·8	42
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk from ration.				
Orange, 4 oz.	0·6	—	6·6	29
	20·6	1·9	31	223
SUPPER				
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk from ration.				
BED-TIME				
1 cup Marmite	0·3	—	—	1
Daily milk ration, 1 pint	18	20	24	348
Daily butter (or vitaminized margarine) ration, 2 oz.	0·2	46·8	—	422
TOTAL	80·8	84·6	120·2	1,564

DIET No. 6A. – More Expensive

Basal Diet for Treatment of Diabetes with Soluble Insulin

Approximately 1,550 Calories with 120 grams Carbohydrate.

INSULIN

	Prot.	Fat	CHO	Calories
BREAKFAST				
Prunes, stewed, 3 oz. or equivalent	0·9	—	12	52
1 egg or Exchange No. 1(*a*) . .	6·2	5·8	0·5	79
Tomato, 3½ oz.	0·9	—	2·1	12
National bread, 1½ oz. or equivalent	3·6	0·6	21·9	107
Tea with milk and cream from ration.				
	11·6	6·4	36·5	250
LUNCH				
Sole or other white fish, 3 oz., or Exchange No. 3 . . .	16·5	1·5	—	79
Salad or other low CHO vegetable, 5 oz.	1·2	—	3	17
Potato, boiled, 2 oz. or equivalent	0·8	—	9·8	42
Fruit as stewed apple, 4 oz., or orange, 2¾ oz.	—	—	4·4	18
Cream from ration.				
	18·5	1·5	17·2	156
TEA				
2 water biscuits or equivalent .	1·6	0·4	10·6	52
Butter from ration.				
Marmite, ⅛ oz.	0·3	—	—	1
Tea with cream from ration.				
	1·9	0·4	10·6	53
INSULIN				
DINNER				
Clear soup, Marmite or Bovril .	0·3	—	—	1
Chicken, average roast and boiled, 3¼ oz., or Exchange No. 2 . .	25·6	8·1	—	175
Salad or other low CHO vegetable, 5 oz.	1·2	—	3	17
Potato, boiled, 2½ oz., or Exchange	1	—	12·2	53
Fruit, as dried apricot, stewed, 3½ oz.	2·1	—	16·1	73
Coffee with milk and cream from ration.				
	30·2	8·1	31·3	319
Daily milk ration, 1 pint . . .	18	20	24	348
Daily butter (or vitaminized margarine) ration, 1½ oz.	0·1	35·1	—	316
Daily 40 per cent cream, 1 oz. . .	0·5	11·9	0·6	111
TOTAL . .	80·8	83·4	120·2	1,558

DIET No. 7

Maintenance Diet for Treatment of Diabetes with Soluble Insulin

Approximately 2,000 Calories with 150 grams Carbohydrate.

	Prot.	Fat	CHO	Calories
INSULIN				
BREAKFAST				
Porridge from 1½ oz. oatmeal or equivalent	5·1	3·7	27·9	165
1 egg or Exchange No. 1(*a*) . .	6·2	5·8	0·5	79
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea with milk from ration.				
	13·7	9·9	43	31
DINNER				
Clear soup, Marmite or Bovril .	0·3	—	—	
Lean meat, 3 oz., or Exchange No. 2	25·5	9·3	—	18
Cabbage or other low carbohydrate vegetable, 7 oz. . . .	1·4	—	2·1	14
Potato, boiled, 2 oz. . . .	0·8	—	9·8	4
or Cereal, ½ oz. as semolina				
Fruit, as prunes, stewed, 3 oz. .	0·9	—	12	5
	28·9	9·3	23·9	29
INSULIN				
HIGH TEA				
White fish, 3 oz., or Exchange No. 3	16·5	1·5	—	7
Melted butter from ration with ¼ oz. chopped parsley . . .	0·3	—	—	
Potato, boiled, 2½ oz. or equivalent	1	—	12·2	5
Salad or low CHO vegetable, 5 oz.	1·2	—	3	1
National bread, 2 oz. . . .	4·8	0·8	29·2	14
Tea with milk from ration.				
	23·8	2·3	44·4	29
SUPPER				
National bread, 1 oz. or equivalent	2·4	0·4	14·6	7
Tea with milk from ration.				
BED-TIME				
1 cup Marmite	0·3	—	—	
Daily milk ration, 1 pint . . .	18	20	24	3
Daily butter (or vitaminized margarine) ration or fat of meat, 3¼ oz. . .	0·3	76	—	6
TOTAL . .	87·4	117·9	149·9	2,0

Should the patient not desire so much butter, he can reduce t amount to 2½ oz. daily, and take 1½ oz. of bacon for breakfast.

DIET No. 7A. – More Expensive

Maintenance Diet for Treatment of Diabetes with Soluble Insulin

Approximately 2,000 Calories with 150 grams Carbohydrate.

INSULIN

	Prot.	Fat	CHO	Calories
BREAKFAST				
Cornflakes, ¾ oz.	1·6	—	18·2	79
Prunes, stewed, 3 oz. or equivalent	0·9	—	12	52
Bacon, 1¼ oz. and 1 egg, or Exchange No. 1(*b*)	9·6	19·9	0·5	219
Tomato, 2 oz.	0·5	—	1·2	7
National bread, 1 oz. or equivalent	2·4	0·4	14·6	72
Tea or coffee with milk from ration.				
	15	20·3	46·5	429
LUNCH				
Sole or other white fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Salad or other low carbohydrate vegetable, 3½ oz.	0·9	—	2·1	12
Potato, boiled, 2½ oz. or equivalent	1	—	12·2	53
Apple, stewed, 4 oz., or orange, 2¾ oz.	—	—	4·4	18
	18·4	1·5	18·7	162
TEA				
3 water biscuits or equivalent	2·4	0·6	15·9	79
Marmite, ⅛ oz.	0·3	—	—	1
Tea with cream from ration.				
	2·7	0·6	15·9	80
INSULIN				
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	1
Chicken, 3¼ oz., or Exchange No. 2	25·6	8·1	—	175
Salad or other low carbohydrate vegetable, 3½ oz.	0·9	—	2·1	12
Potato, boiled, 2½ oz. or equivalent	1	—	12·2	53
Fruit as dried apricot, stewed, 3 oz.	1·8	—	13·8	62
3 water biscuits or equivalent	2·4	0·6	15·9	79
Cheddar cheese, ¾ oz.	5	7	—	83
Coffee with milk and cream from ration.				
	37	15·7	44	465
Daily milk ration, 1 pint	18	20	24	348
Daily butter (or vitaminized margarine) ration, 1¼ oz.	0·1	29·2	—	263
Daily 40 per cent cream, 2¼ oz.	1·1	26·8	1·8	251
TOTAL	92·3	114·1	150·4	1,998

DIET No. 8

Maintenance Diet for Treatment of Diabetes with Zin Protamine Insulin

Approximately 2,000 Calories with 150 grams Carbohydrate.

ZINC PROTAMINE INSULIN BEFORE BREAKFAST

	Prot.	Fat	CHO	Calor
BREAKFAST				
Porridge from ¾ oz. oatmeal or equivalent	2·5	1·8	13·9	8
1 egg or Exchange No. 1(*a*) . .	6·2	5·8	0·5	7
National bread, 1 oz. . . .	2·4	0·4	14·6	7
Milk from ration for tea and porridge.				
	11·1	8	29	2.
DINNER				
Clear soup, Marmite or Bovril	0·3	—	—	
Chicken, 3¼ oz., or Exchange No. 2	25·6	8·1	—	1
Salad or other low carbohydrate vegetable, 4 oz. . . .	1	—	2·4	
Potato, boiled, 2 oz. or equivalent	0·8	—	9·8	
Fruit as prunes, stewed, 4 oz. .	1·2	—	16	
Curds with milk from ration.				
	28·9	8·1	28·2	3
MID-AFTERNOON				
National bread, 1½ oz. or equivalent	3·6	0·6	21·9	1
Marmite, ⅛ oz.	0·3	—	—	
Tea with milk from ration.				
	3·9	0·6	21·9	1
HIGH TEA				
White fish, 3 oz., or Exchange No. 3	16·5	1·5	—	
National bread, 2 oz. or equivalent	4·8	0·8	29·2	1
Salad or other low carbohydrate vegetable, 3½ oz. . . .	0·9	—	2·1	
Tea with milk from ration.				
	22·2	2·3	31·3	2
BED-TIME				
3 water biscuits or equivalent .	2·4	0·6	15·9	
Milk from ration.				
Daily milk ration, 1 pint . . .	18	20	24	3
Daily butter (or vitaminized margarine) ration, 3⅓ oz.	0·3	78	—	7
TOTAL . . .	86·8	117·6	150·3	2,0

N.B. – Should the patient not desire so much butter, he can red the amount to 2⅓ oz. daily and take 1¾ oz. bacon for breakfast.

DIET No. 8A.—More Expensive

Maintenance Diet for Treatment of Diabetes with Zinc Protamine Insulin

Approximately 2,000 Calories with 150 grams Carbohydrate.

Zinc Protamine Insulin before Breakfast

	Prot.	Fat	CHO	Calories
Breakfast				
Fruit, as orange, 4 oz.	0·6	—	6·6	29
2 eggs or Exchange No. 1(*a*)	12·4	11·6	1	158
National bread, 1½ oz. or equivalent	3·6	0·6	21·9	107
Tea with cream or milk from ration.				
	16·6	12·2	29·5	294
Lunch				
Sole or other white fish, 3 oz., or Exchange No. 3	16·5	1·5	—	79
Salad or other low carbohydrate vegetable, 5 oz.	1·2	—	3	17
Potato, 2½ oz., or exchange	1	—	12·2	53
Fruit as dried apricot, stewed, 3 oz.	1·8	—	13·8	62
Curds with milk from ration.				
	20·5	1·5	29	211
Tea				
National bread, 1¼ oz.	3	0·5	18·2	89
Marmite, ⅛ oz.	0·3	—	—	1
Tomato, lettuce, cress as sandwich, 2½ oz.	0·6	—	1·5	8
Tea with cream or milk from ration.				
	3·9	0·5	19·7	98
Dinner				
Clear soup, Marmite or Bovril	0·3	—	—	1
Chicken, roast, 3¼ oz., or Exchange No. 2	25·6	8·1	—	175
Salad or other low carbohydrate vegetable, 4 oz.	1	—	2·4	14
Potato, boiled, 2½ oz. or equivalent	1	—	12·2	58
Fruit as prunes, stewed, 4 oz.	1·2	—	16	69
Whipped cream from ration.				
Coffee with milk from ration.				
	29·1	8·1	30·6	312
Bed-time				
Eating apple, 4½ oz.	0·4	—	10·8	45
1 water biscuit	0·8	0·2	5·8	26
Daily milk ration, 1 pint	18	20	24	348
Daily butter (or vitaminized margarine) ration, 2¼ oz.	0·2	52·6	—	474
Daily 40 per cent cream, 2¼ oz.	0·9	20·8	1	195
Total	90·4	115·9	149·9	2,003

DIET No. 9

Light Diabetic Diet

Approximately 1,500 Calories with 150 grams Carbohydrate.

INSULIN (SOLUBLE)

	Prot.	Fat	CHO	Calori
BREAKFAST				
Strained porridge, 3 tablespoons or equivalent	2·5	1·8	13·9	8
White crustless bread, toasted, 1 oz. (weigh before toasting)	2·3	0·2	15·6	7
1 egg, poached or scrambled	7·2	6·6	0·6	9
Milk for tea and porridge from ration.				
	12	8·6	30·1	2
FORENOON				
Cup of Marmite	0·3	—	—	
2 water biscuits or equivalent	1·6	0·4	10·6	
	1·9	0·4	10·6	
DINNER				
Chicken, boiled, 2¼ oz., or Exchange No. 3	16·6	6·5	—	1
Carrot, boiled, sieved, 2 oz.	0·4	—	2·2	
Crustless white bread, 1 oz.	2·3	0·2	15·6	
Apple, stewed, 4 oz. or equivalent	—	—	4·4	
Serve with milk from ration.				
	19·3	6·7	22·2	2
INSULIN (SOLUBLE)				
TEA				
White fish, steamed, 3 oz., or Exchange No. 3	16·5	1·5	—	
Parsley, chopped, ¼ oz.	0·3	—	—	
White crustless bread, 2 oz.	4·6	0·4	31·2	1
Butter (or vitaminized margarine) from ration.				
Milk for tea and fish from ration.				
	21·4	1·9	31·2	
SUPPER				
Orange juice, 4½ oz.	0·9	—	10·8	
Glucose, ½ oz.	—	—	13·5	
Water.				
	0·9	—	24·3	
BED-TIME				
Benger's or fine cereal, ⅓ oz.	—	—	8·1	
Milk from ration.				
Daily milk ration, 1 pint	18	20	24	
Daily butter (or vitaminized margarine) ration, 1¼ oz.	0·1	29·2	—	
TOTAL	73·6	66·8	150·5	1,

DIET No. 10

First Stage Peptic Ulcer Diet

Approximately 2,000 Calories.

On Waking 5 oz. milk.
1 oz. cream.

Breakfast, 9 a.m. Flour of meal or strained porridge (1 teacup).
5 oz. milk.
1 oz. cream.
1 rusk or crisp toast buttered when cold.
or Scrambled egg.
2 rusks or 2 thin slices toast buttered cold.
1 cup milk tea.

11 a.m. 5 oz. milk. 1 oz. cream.
or Milk soup or proprietary food (*e.g.* Benger's or Farex).
1 rusk or crisp toast buttered when cold.

Lunch, 1 p.m. Small helping of fish custard, soufflé or cream.
or Chicken or rabbit soufflé or cream.
or Brains or sweetbread or tripe (well cooked in milk, no onion).
Simple white sauce (no parsley or seasoning).
Purée of spinach, carrot or tomato.
Later – 1 tablespoon mashed potato.
Orange jelly or junket or prune or apple whip. Glucose.
Cream, 1 oz.

3 p.m. Same as 11 o'clock.

Tea, 5 p.m. 5 oz. milk or 1 cup freshly infused, milky China tea. Sugar. Cream.
1 egg – poached, lightly boiled or scrambled.
1 thin slice crisp toast buttered cold, or 2 tea rusks.
Butter (or vitaminized margarine); honey, jelly or treacle.
Later – add small sponge or Madeira cake (no fruit).

Evening Meal, 7 p.m. Well cooked milk pudding with egg and cream.
or Steamed, baked, or pouring custard with cream.
Purée of fruit as apple, prunes, apricot, pear or banana. Sugar.

Bed-time. Same as 11 a.m.
If awake during night, 5 oz. milk.

Between feeds: Strained juice of 2 oranges diluted with water.

Intermediate Stage

Same as above – only quantities increased.
Small helping of tender beef or mutton allowed and fish, chicken or rabbit need not be made into purées or creams.

For Post-Ulcer Regime, see Bland Diet Instruction, pp. 264 to 266.

DIET No. 11

Bland Diet suitable for Functional Achlorhydric Dyspepsi or Chronic Gastritis with Hypochlorhydria or Achlorhydri

Early Morning	Orange or tomato juice.
Breakfast	Cornflakes or shredded wheat. 1 egg, poached, and small piece of grilled crisp bacon. or Average helping white fish steamed or baked. Crisp thin toast – buttered sparingly when cold. Honey or jelly marmalade. 1 cup freshly infused weak tea. Milk. Sugar.
11 a.m.	Cup of Marmite. A finger of crisp toast.
Dinner	Clear meat soup or beef tea. Tender beef or mutton, rabbit or chicken finely minced average helping. Baked or mashed potato. If flatulence, omit, and giv crisp toast or a little rice, flaky and dry. 1 tablespoon sieved vegetable, flower of cauliflower c sieved tomato. Milk pudding or custard made with egg, or curds c fruit jelly, or baked apple or puréed apple, apricot c prunes.
Tea	1 cup freshly infused tea. Milk. Sugar. Well toasted bread or thin bread baked in oven, or cris biscuit. Very little butter (or vitaminized margarine). Jelly or honey. Later – small piece sponge cake.
Supper	White fish, sweetbread or tripe – average helping. Crisp toast buttered very sparingly when cold. Cup of milk.
Bed-time	Cup of warm milk, or Benger's or Allenbury's c Horlick's or cocoa made with milk.

For general instructions regarding indigestible articles of food a physiological methods of eating, see pp. 264 to 265.

DIET No. 12

Lacto-vegetarian Diet – Moderate Protein and High Vitami

Early Morning	Orange juice.
Breakfast	Porridge, cornflakes or any crisp breakfast cereal. Cream. Wholemeal bread (toasted if desired), 2 thin slices. Butter (or vitaminized margarine). Honey or marmalade. 1 cup freshly made tea with milk from ration.
11 a.m.	Glass of buttermilk or cup of Marmite.

Dinner	Milk soup with sieved vegetable and Marmite added. Macaroni and cheese. or Cauliflower au grâtin. or Baked tomato with breadcrumbs and cheese. Always salad in season with grated raw carrot or a large helping of vegetable as carrot, flower of cauliflower, spinach. Potato, mashed or baked in skin. Fresh or stewed fruit. Curds if liked or milk from ration to drink.
Afternoon	Glass of water.
Tea	St. Ivel cheese, ¾ oz., or egg dish. Tender lettuce, tomato, cress, etc., a large helping. Crisp toast or wholemeal bread – butter (or vitaminized margarine). Weak tea with milk from ration.
Supper	Glass of buttermilk. Wholemeal bread and butter (or vitaminized margarine). Fruit as at dinner.
Bed-time	Glass of water.
Daily Ration	Milk, 1 pint. Buttermilk, 1 pint.

DIET No. 13

Chronic Pancreatic Deficiency (Initial Treatment)

Breakfast	Small helping of any cereal with stewed fruit and sugar. Crisp toast; syrup, honey, or marmalade jelly. Tea or coffee with skimmed milk.
Mid-morning	Orange or apple or fruit drink.
Lunch	Marmite, clear soup or skimmed milk vegetable soup. Small helping of white fish (boiled or steamed), chicken or very lean meat. Sieved green or yellow vegetables. Small serving puréed potato. Fruit, fresh or stewed, or fruit jelly or skimmed milk jelly.
Tea	Rusks or crisp toast; jam or honey. Tea with skimmed milk.
Dinner	As lunch.
Supper	6 oz. of peptonized milk or Benger's food made with skimmed milk.

Note. – ½ pint of skimmed milk and ½ pint of peptonized milk must be taken daily.

For Maintenance Diet, see p. 285.

DIET No. 14

Tropical Sprue and Idiopathic Steatorrhœa

1. *High Protein Meat Diet* (Hamilton Fairley)

DIET No. 1 (Caloric value = 770)

8 A.M.	Underdone beef, 3 oz.; rusks, ¾ oz.; juice of ½ orange; and glucose, 2 drachms.
12 NOON	Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 3 oz.; rusks, ¾ oz.; juice of ½ orange; and glucose, 1 drachm.
6 P.M.	The same as at 12 noon.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·2.

Note. – When patients are very ill, two-hourly feeds of meat or beef-juice can be substituted.

DIET No. 2 (Caloric value = 1,280).

8 A.M.	Underdone beef, 5 oz.; rusks, 1 oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, 2 drachms.
12 NOON	Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 5 oz.; rusks, 1 oz.; juice of 1 orange + glucose, 2 drachms.
4 P.M.	Tea, 10 oz.; milk, 2 oz.
7 P.M.	The same as at 12 noon + calves-foot jelly, 2 oz.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·0.

DIET No. 3 (Caloric value = 1,820)

6 A.M.	Tea, 10 oz.; milk, 2 oz.
8 A.M.	Underdone beef, 6 oz.; rusks, 1½ oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, 2 drachms.
10 A.M.	1 baked apple; custard, 1 oz.
12 NOON	Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 6 oz.; calves-foot jelly, 2 oz.; rusks, 1½ oz.; juice of 1 orange + glucose, 2 drachms.
4 P.M.	Tea, 10 oz.; milk, 2 oz.; baked apple, 1 oz.; custard, 1 oz.
7 P.M.	The same as at 12 noon.

Protein : fat : carbohydrate = 1·0 : 0·32 : 1·3.

DIET No. 4 (Caloric value = 2,200)

6 A.M.	Tea, 10 oz.; milk, 2 oz.
8 A.M.	Underdone beef, 7 oz.; rusks, 1½ oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, 2 drachms.
10 A.M.	1 baked apple + custard, 2 oz.
12 NOON	Soup, 5 oz. + liver extract (= ⅝ lb.); underdone beef, 7 oz.; calves-foot jelly, 2 oz.; rusks, 3 oz.; juice of 1 orange + glucose, 2 drachms.

4 P.M. Tea, 10 oz.; milk, 2 oz.; 1 baked apple; custard, 3 oz.

7 P.M. The same as at 12 noon, but only 1½ oz. of rusks allowed.

Protein : fat : carbohydrate = 1·0 : 0·34 : 1·3.

Diet No. 5 (Caloric value = 3,020)

6 A.M. Tea, 10 oz.; milk, 2 oz.; glucose, 2 drachms; rusks, 1½ oz.; butter (or vitaminized margarine), 1 drachm; 1 scraped ripe apple or 1 fully ripe Canary banana (yellow ends).

8 A.M. Underdone beef, 7 oz.; rusks, 3 oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, ½ oz.; honey, 2 drachms; butter (or vitaminized margarine), 1 drachm.

10 A.M. 1 baked apple; custard, 3 oz.

12 NOON Soup, 5 oz. + liver extract (= ⅝ lb.); underdone beef, 7 oz.; calves-foot jelly, 2 oz.; rusks, 1½ oz.; juice of 1 orange + glucose, ½ oz.

4 P.M. Tea, 10 oz.; milk, 2 oz.; glucose, 2 drachms; rusks, 3 oz.; baked apple, 1 oz.; custard, 3 oz. (egg boiled or poached sometimes substituted); honey, 2 drachms.

7 P.M. The same as at 12 noon.

Protein : fat : carbohydrate = 1·0 : 0·36 : 2·0.

DIET No. 15

2. *High Protein Milk* (*Sprulac*) *Diet* (Hamilton Fairley)

Diet No. 1 (Caloric value = 697)

1 oz. of Sprulac made up to 8 oz. with water every 2½ hours for six feeds (7.30 a.m. to 7.30 p.m.).

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·3.

Diet No. 2 (Caloric value = 1,117)

1½ oz. of Sprulac made up to 12 oz. with water every 2½ hours for six feeds (7.30 a.m. to 7.30 p.m.). The juice of 1 orange; calves-foot jelly, 1 oz.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·4.

Diet No. 3 (Caloric value = 1,536)

2 oz. of Sprulac made up with 16 oz. water are given every 2½ hours for six feeds (7.30 a.m. to 7.30 p.m.). The juice of 2 oranges; calves-foot jelly, 2 oz.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·5.

Diet No. 4 (Caloric value = 2,001)

Sprulac – as Diet No. 3 above.

Juice of 2 oranges; calves-foot jelly, 3 oz.; 2 baked apples; custard, 2 oz.; underdone beef, 4 oz.; ½ rusk, ¾ oz.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·5.

Diet No. 5 (Caloric value = 2,516)

Sprulac – as Diet No. 3 above.
Juice of 2 oranges; calves-foot jelly, 3 oz.; 2 baked apples; custard, 2 oz.; 2 rusks, 3 oz.; underdone beef, 10 oz.

Protein : fat : carbohydrate = 1·0 : 0·3 : 1·4.

As far as possible the Sprulac feed should be given at intervals of two and a half hours, and the other constituents mainly taken at 9 a.m., 1 p.m., and 5 p.m. A certain latitude is permissible in this respect and in some patients, especially when very debilitated, the feeds may be given in smaller quantities more frequently and during the night. The rapidity with which the diets are changed is determined by the progress of the patient as indicated by the disappearance of intestinal flatulence and abdominal distension, and the character of the stools. When progress is satisfactory, the latter decrease in bulk and number, become progressively more solid, and lose their gaseous features. Chemical analysis also shows a decrease in the total fæcal fat. Not uncommonly it is possible to pass from No. 1 to No. 5 Diet within a period of three or four weeks.

During convalescence a more liberal dietary is permitted.

DIET No. 16

Sprue: Convalescent Stage

BREAKFAST (8 to 9 A.M.)	Lightly boiled or poached eggs or underdone lean chop or steak or filleted, boiled or steamed fish – whiting, sole, plaice, haddock; thin toast, and butter (or vitaminized margarine) in moderation; weak tea; stewed fruit; apples, rhubarb; honey or jam in small quantity.
11 A.M.	½ pint of milk, if desired, and if it agrees.
LUNCH	Chicken or vegetable soup; underdone grilled steak or roast or boiled chicken or liver cooked in various ways or cold beef or mutton, but fat not to be eaten. Vegetables: spinach, marrow, cauliflower, French beans, celery, young peas, boiled onions; salad of lettuce and tomato; boiled potatoes in small quantity. Custard, junket, milk jellies, fruit jelly or baked apples. Small quantity of cream allowed. Fresh fruit such as oranges, Canary bananas, pears, peaches, grapes, raspberries, strawberries, melons, grapefruit. Rusks or toast allowed.
TEA (4 P.M.)	Weak China tea. Madeira cake or sponge cake, dry toast and butter (or vitaminized margarine), Marie or water biscuits.
DINNER (7 P.M.)	Chicken, rabbit, brains, sweetbread, tripe or cold lean meat; lettuce and tomato salad or other vegetables mentioned, but no potatoes; custard, junket, baked apple, stewed fruit or fruit in jelly, and a small quantity of fresh cream; rusks or toast and butter (or vitaminized margarine) allowed.

GENERAL ADVICE

1. The 7 o'clock meal should be the lightest in the day. The patient must watch his diet carefully, and if he has any idiosyncrasy, then he must use common sense and avoid any article of food that disagrees.

2. Vegetables must be well cooked, and boiled, not fried.

Meat should be underdone.

Sugar and starchy foods like boiled potatoes, milk puddings made from rice, sago, tapioca and semolina, also cereals and bread, should be taken at first in moderation, and the quantity not increased till the patient is sure that they agree.

Milk fat is the best tolerated of all fat in sprue, but milk, cream and butter (or vitaminized margarine) are allowed only in very moderate amounts at first. They may be increased gradually in quantity. Patients are also instructed to cut out all obvious meat fat, whether mutton or beef.

ARTICLES TO BE AVOIDED

The following substances must be avoided:

Overdone and twice-cooked meat and articles fried or cooked in fat; condiments, like pepper, mustard, chillies, sauces, chutneys, curries and spiced food; game; duck and fat fish, such as salmon, trout, mackerel and herrings; fried bread, grease, fat, salad oil dressings and sauces of all kinds, suet puddings, cakes with icing, raisins and pastry; sweets and chocolates; and alcoholic drinks and mineral waters.

DIET No. 17

Atonic Constipation in a person of Normal or less than Normal Weight

High Vitamin B. High Fat. High Residue.

EARLY A.M.	Glass of water.
BREAKFAST	Stewed prunes or figs or baked or raw apple with 2 tablespoons of Kellogg's All Bran. or Porridge, creamy milk and 1 tablespoonful of Bemax. Crisp toast (buttered liberally when cold) or stale wholemeal bread or oatcake. Butter plentifully. Marmalade or treacle. Weak tea or coffee – milk and sugar as desired.
11 A.M.	Glass of buttermilk or cup of Marmite.
DINNER	Vegetable soup as often as possible. Small helping meat or fish (not fried and not twice cooked). Large helping boiled onion or cabbage or other green vegetable. Small potato – baked in skin. Always stewed or fresh fruit. Glass of water.
TEA	Small helping of meat, fish or egg. Always a helping of salad in season, or vegetable. Brown bread or wholemeal scone. Butter liberally. Gingerbread made with treacle, raisins and nuts.

SUPPER Glass of buttermilk preferably.
Brown bread and butter (or vitaminized margarine).
Treacle.

BED-TIME Glass of water. Raw apple.

Restrict foods rich in carbohydrate because of their tendency to produce flatulence, *e.g.* pastries, rich puddings, new and white bread, scones, cookies and rolls.

TAKE IN GENEROUS HELPINGS

1. Vegetables selected from lettuce, spinach, sprouts, cabbage, cauliflower, onion, tomato, parsnip, carrot, asparagus and celery.
2. Fruits, fresh, stewed or preserved. Apples, cherries, grapes, pears, peaches, pineapple, plums, oranges, rhubarb. Also dried fruits as figs, dates, prunes and raisins. Nuts are helpful.
3. Fats such as butter (or vitaminized margarine), cream, olive oil, bacon fat and dripping unless patient is overweight.
4. Water – in liberal quantity – between meals.

DIET No. 17A – More Expensive

Atonic Constipation in a Person of Normal or Less than Normal Weight

High Vitamin B. High Fat. High Residue.

EARLY A.M. Juice 1 orange.
,, ½ lemon. } Water to ½ pint.
2 tablespoons lactose.

BREAKFAST ½ grapefruit with 1 tablespoon lactose or stewed prunes, figs or apples with 2 tablespoonfuls of Kellogg's All Bran.
Porridge made from coarse oatmeal, with Bemax, 1 tablespoon.
Cream.
Wholemeal bread – butter liberally.
Honey, marmalade or treacle.
Coffee.
Egg or bacon if wished, but above foods are more important.

11 A.M. Glass of buttermilk preferably or glass of water and fresh fruit.

LUNCH Egg or fish dish.
Raw salad – olive oil dressing.
Wholemeal bread. Butter liberally.
Prunes, apricots, or figs. Cream.
Glass of buttermilk preferably or coffee.

TEA Cup of weak tea.
Wholemeal bread made into sandwich with tomato, lettuce or Marmite. Butter liberally.
or Nuts and cream cheese sandwich.
Piece of gingerbread made with treacle, raisins and nuts.

Dinner	Vegetable soup – milk, vegetable and a little cream. Add Marmite. Average serving meat or fish. Potato baked in skin. Serve with butter (or vitaminized margarine). Large helping of vegetable, especially cabbage and boiled onion. In season – always salad of lettuce, tomato, cress, grated raw carrot. Salad dressing. Any simple sweet. Always fruit, raw or stewed. Cream.
Bed-time	Raw apple. Glass of water.

Restrict foods rich in carbohydrate because of their tendency to produce flatulence, *e.g.* pastries, rich puddings, new and white bread, scones, cookies and rolls.

Take in Generous Helpings

1. Vegetables selected from lettuce, spinach, sprouts, cabbage, cauliflower, onion, tomato, parsnip, carrot, asparagus and celery.
2. Fruits – fresh, stewed or preserved. Apples, cherries, grapes, pears, peaches, pineapple, plums, oranges, rhubarb. Also dried fruits as figs, dates, prunes and raisins. Nuts are helpful.
3. Fats such as butter (or vitaminized margarine), cream, olive oil, bacon fat and dripping unless patient is overweight.
4. Water – in liberal quantity – between meals.

DIET No. 18

Atonic Constipation + Obesity

Approximately 1,200 Calories.

Early Morning	Glass of water.
Breakfast	Stewed prunes or figs – no sugar. Saccharin if desired. 1 egg. Wholemeal bread – 1 oz. – may be toasted and buttered when cold. Butter (or vitaminized margarine) from ration. Tea with milk from ration. No sugar.
Forenoon	Buttermilk from ration, or cup of Marmite.
Dinner	Clear soup or cup of Marmite. Lean meat, or tripe, chicken, rabbit, or white fish not fried – average helping. Large helping green vegetable. No potato. 1 apple, raw, baked or stewed without sugar; or orange or fruit jelly.
Afternoon	Glass of water.
Tea	White fish – not fried – small helping. Always salad in season or green vegetable or tomato. Wholemeal bread, 1½ oz. Butter (or vitaminized margarine) from ration. Tea with milk from ration.

Supper 1 cup tea with milk from ration.
Wholemeal bread, 1 oz.
Butter (or vitaminized margarine) from ration.
Raw apple.

Rations for Day. Milk, ½ pint.
Buttermilk, ½ pint.
Butter (or vitaminized margarine), 1 oz.

DIET No. 19

Spastic Constipation

Low Residue. High Vitamin. Bland Diet.

Early Morning Glass of water, hot or cold.

Breakfast Cup of weak, freshly infused tea. Milk or preferably cream.
Well boiled and strained porridge. Cream or milk.
Egg lightly cooked (not fried).
or Fat bacon grilled.
Cold crisp toast. Plenty of butter (or vitaminized margarine).
Black treacle or golden syrup.

11 a.m. Glass of buttermilk,
or Cup of Marmite and plain biscuit.

Dinner Average helping of tender meat,
or Steamed fish or rabbit or tripe (no onion).
Small helping sieved vegetable.
Potatoes baked in skin or mashed. Do not eat skin.
Curds or custard or milk pudding.
Baked or stewed apple, sieved apricots or prunes, or fruit jelly.

Afternoon Glass of buttermilk,
or Strained orange juice.

Tea White bread crisply toasted or fine brown bread as Hovis.
Make sandwich with Marmite or St. Ivel cheese.
Simple cake if desired.
Cup of weak, freshly infused tea.

Supper If not hungry, Horlick's, Benger's or cocoa or any milk food, or, preferably,
Fish or egg – poached, boiled or scrambled, or ham (nothing fried).
Well-made toast or fine brown bread.
Treacle or golden syrup.
Mashed banana.

Bed-time Glass of water.

Take Plentifully

Orange juice or strained tomato juice. Marmite.
Sieved vegetables or mashed carrot or flower of cauliflower.

AVOID

Much fluid with meals but take plentifully between meals.
All coarse-fibred meat.
All fried and highly seasoned food and twice-cooked meats.
All coarse cereals and pastries.
All heavy puddings.
All fruit with seeds and husks.
All coarse vegetables as cabbage, sprouts, stalk of cauliflower, savoy, peas, beans, lentils, leeks, onions, celery, raw salads.
Extremes of temperatures in foods.

DIET No. 19A. – More Expensive

Spastic Constipation

Low Residue. High Vitamin. Bland Diet.

EARLY MORNING	Strained juice of 1 orange. 1 level tablespoon lactose. Water.
BREAKFAST	1 cup freshly infused China tea. Cream. Cornflakes and sieved prunes or baked apple (avoiding skin and core). Cream. Bemax, 1 tablespoon. Fillet of fish steamed or done in oven, or 2 rashers bacon (grilled) and poached egg. Crisp toast or fine brown bread (stale) or rusks. Butter (or vitaminized margarine). Honey or jelly of marmalade.
11 A.M.	Repeat fruit drink or orange and egg flip or cup of Marmite.
LUNCH	½ wineglass strained tomato juice or Heinz bottled tomato juice. White fish, if not taken at breakfast, or steamed liver or tender, lean meat minced and broiled or rabbit or chicken soufflé or cream or sweetbread or tripe. Old potato, mashed or baked in skin (no new waxy potato). Vegetable purée. Spinach is best or carrot, flower of cauliflower, marrow, asparagus tips and later young French beans. Curds, custard or milk jelly. Serve with cream. Always fruit juice or stewed fruit (no pips, skins or seeds), or fruit purées, creams, whips, jellies or soufflés.
TEA	Freshly infused China tea. Thin slice fine brown bread and butter (or vitaminized margarine). Honey, bramble or apple jelly. Sponge cake (no fruit, jams, nuts, etc.).
DINNER	Cream soup, *e.g.* asparagus, celery or cream of barley, or strained tomato juice, or milk with Marmite thickened with 1 teaspoon cornflour, arrowroot or potato flour.

Fish or meat as at lunch.
Vegetable as above.
Sweet as above.
Breakfast biscuit or fairy toast (no digestive biscuits).
Cream cheese.

10 P.M. Repeat fruit drink.

TAKE PLENTIFULLY

Cream, butter (or vitaminized margarine), lactose, honey.
Buttermilk is beneficial in some cases, but not in all.
Orange juice or strained tomato juice, and sieved fruits.
Sieved vegetables or mashed carrot or head of cauliflower.
Marmite.

AVOID

Much fluid with meals but take plentifully between meals.
All coarse-fibred meat.
Fried and highly seasoned food and twice-cooked meats.
Coarse cereals and pastries.
Heavy puddings.
Fruit with seeds and husks.
Coarse vegetables as cabbage, sprouts, stalk of cauliflower, savoy peas, beans, lentils, leeks, onions, celery, raw salads.
Extremes of temperatures in foods.

DIET No. 20

Acute Severe Hepatitis (Acute Yellow Atrophy)

(For Details of Initial Treatment, see p. 301.)

1. Acute severe hepatitis (recovering).
2. Subacute and mild hepatitis.
3. Obstructive jaundice.

High CHO. Low Fat. High Casein Diet.

Approximately: Prot. 80. Fat 20. CHO 400. Calories 2,100.

1ST STAGE DIET

EARLY MORNING Glass of water or fruit drink, hot or cold.

BREAKFAST 5 to 6 tablespoonfuls porridge (strained), or averag serving cornflakes or other breakfast cereal.
Milk from ration.
Bread, 1 slice, toasted.
Jelly marmalade – no butter.
Weak tea with milk and sugar from ration.

FORENOON Fruit drink.

DINNER Lean meat or white fish, not fried, small serving.
or Exchange No. 3 (see p. 446).
Potato, 4 oz.
Small serving sieved vegetables.
Cereal pudding made with milk from ration.
Fruit, fresh or stewed with sugar.

Tea Bread, 1½ thin slices – may be toasted.
or Exchange No. 4 (see p. 447).
Jelly or honey.
Weak tea with milk and sugar from ration.

Supper Bread, 2 thin slices – may be toasted.
or Exchange No. 4 (see p. 447).
Jelly or honey.
Weak tea with milk and sugar from ration.
Fruit may be taken at this meal instead of at dinner.

Bed-time Fruit drink.

Daily Rations 1 pint fruit drink made with 100 grams glucose to be taken throughout the day.
Milk, whole, ½ pint.
Milk, skim, 2 pints or 1 pint special milk (double strength Household Milk).
Jelly or honey, 1½ oz.
Sugar, 2 oz.
No butter or margarine.

General Instructions

Eat slowly and chew thoroughly.

Avoid

All fried and fatty foods, all foods made with fat, oils, cream, cheese, egg yolks.
Duck, goose, salmon, bacon, herring.
All pastries, Yorkshire and suet puddings.
Chocolate, cocoa, strong tea and coffee.
Condiments, spices and alcoholic beverages.

Take Freely

Fresh fruits and vegetables which should be sieved.
Honey, jam and sugar.
Water and fruit drinks.

DIET No. 20A. – More Expensive

Acute Severe Hepatitis (Acute Yellow Atrophy)

(For Details of Initial Treatment, see p. 301.)

1. Acute severe hepatitis (recovering).
2. Subacute and mild hepatitis.
3. Obstructive jaundice.

High CHO. Low Fat. High Casein Diet.

Approximately: Prot. 80. Fat 20. CHO 400. Calories 2,100.

1st Stage Diet

Early Morning Glass of water or fruit drink, hot or cold.

BREAKFAST Average serving cornflakes or other breakfast cere
with 2 tablespoons syrup.
Fruit, *e.g.* stewed prunes or mashed banana, averag
serving.
Bread, 2 thin slices as crisp toast.
Jelly marmalade or honey.
Weak tea with milk and sugar from ration.

FORENOON Juice 1 orange } Glass water, hot or cold.
,, ½ lemon. } Sugar from ration.

LUNCH Small serving steamed white fish, tripe, chicken
rabbit.
Coating sauce without butter, using cornflour and mi
from ration.
Potato, 4 oz., boiled or baked.
Small serving sieved vegetables.
Average serving stewed or raw ripe fruit.
Few boiled sweets.

MID-AFTERNOON Fruit drink as above.

TEA Bread, 2 thin slices or equivalent.
Jelly, honey or syrup.
Weak tea with milk and sugar from ration.

DINNER Small serving tripe, chicken, rabbit or fish.
Coating sauce, as above.
Potato, 3 oz., or Exchange No. 4 (see p. 447).
Lemon sago or other high CHO, low fat sweet.

BED-TIME Fruit drink as above.

DAILY RATIONS Milk, whole, ½ pint.
Milk, skim, 2 pints or 1 pint special milk (doub
strength Household Milk).
Butter (or vitaminized margarine), ¼ oz.
Sugar, 2 oz.
Jelly, honey or syrup, 1½ oz.

GENERAL INSTRUCTIONS

Eat slowly and chew thoroughly.

AVOID

All fried and fatty foods, all foods made with fat, oils, cream, chees
egg yolks.
Duck, goose, salmon, bacon, herring.
All pastries, Yorkshire and suet puddings.
Chocolate, cocoa, strong tea and coffee.
Condiments, spices and alcoholic beverages.

TAKE FREELY

Fresh fruits and vegetables which should be sieved.
Honey, jam and sugar.
Water and fruit drinks.

DIET No. 21

1. Acute Severe Hepatitis (recovering)
2. Subacute and Mild Hepatitis (recovering)
3. Obstructive Jaundice (recovering)

High CHO. Low Fat. High Protein Diet.

Approximately: Prot. 120. Fat 45. CHO 400. Calories 2,500.

2nd Stage Diet

Early Morning	Glass of water or fruit drink, hot or cold.
Breakfast	6 to 8 tablespoonfuls porridge. Milk from ration. Bread, 2 slices – may be toasted. Butter from ration. Jelly marmalade. Tea with milk and sugar from ration.
Forenoon	Glass of water or fruit drink, hot or cold.
Dinner	Lean meat, 3 oz. (not fried), or Exchange No. 2 (see p. 446). Potato, 5 oz. Average serving sieved vegetable. Cereal pudding made with milk from ration. Fruit, fresh or stewed, with sugar, now or at supper.
Tea	Bread, 3 slices – may be toasted, or equivalent. Butter from ration. Jam, jelly or honey. Weak tea with milk and sugar from ration.
Supper	White fish, 3 oz. (not fried), or Exchange No. 3 (see p. 446). Bread, 3½ oz. or equivalent. Butter from ration. Jam, jelly or honey. Weak tea or coffee with milk and sugar from ration.
Bed-time	Glass of water, hot or cold.
Daily Rations	Milk, whole, ½ pint. Milk, skim, 2 pints or 1 pint special milk (double strength Household Milk). Butter (or vitaminized margarine), ½ oz. Sugar, 2 oz. Jam, honey or syrup, 2 oz.

General Instructions

Eat slowly and chew thoroughly.

Avoid

Large meals and indigestible articles of food.
All fried and fatty foods, all foods made with fat, oils, cream, egg yolks.
Duck, goose, salmon, bacon, herring.
All pastries, Yorkshire and suet puddings.
Chocolate, cocoa, strong tea and coffee.
Condiments, spices and alcoholic beverages.

TAKE FREELY

Fresh fruits and vegetables which should be sieved.
Honey, jam and sugar.
Water and fruit drinks.

DIET No. 21A. – More Expensive

1. Acute Severe Hepatitis (recovering)

2. Subacute and Mild Hepatitis (recovering)

3. Obstructive Jaundice (recovering)

High CHO. Low Fat. High Protein Diet.

Approximately: Prot. 120. Fat 45. CHO 400. Calories 2,500.

2ND STAGE DIET

EARLY MORNING	Glass of water, hot or cold.
BREAKFAST	5 to 6 tablespoonfuls porridge, or average serving cor flakes or other breakfast cereal with 2 tablespoc syrup, or Fruit, *e.g.* stewed prunes or mashed banana, avera serving. Fish, small serving, or egg, boiled or poached. Bread, 2 thin slices, as crisp toast. Butter from ratic Jelly marmalade or honey. Weak tea with milk and sugar from ration.
FORENOON	Juice 1 orange } Glass hot water. ,, ½ lemon. } Sugar from ration.
LUNCH	Small serving, 2 oz., steamed white fish, tripe, chick or rabbit. Coating sauce without butter, using cornflour and m from ration. Potato, 5 oz., boiled or baked. Average serving sieved vegetable. Average serving stewed, or raw, ripe fruit. Few boiled sweets.
MID-AFTERNOON	Fruit drink as above.
TEA	Bread, 1½ thin slices – may be toasted. Jelly, honey or syrup. Weak tea with milk and sugar from ration.
DINNER	Average serving (3 oz.) lean meat, liver, fish, chicken rabbit – not fried. Crisp toast, 2½ slices or equivalent. Small serving vegetable (sieved). Average serving fruit, stewed or raw. Biscuit (sweet). Weak coffee with milk and sugar from ration.
BED-TIME	Fruit drink as above.

DAILY RATIONS
Milk, whole, ½ pint.
Milk, skim, 2 pints or 1 pint special milk (double strength Household Milk).
Butter (or vitaminized margarine), ½ oz.
Sugar, 2 oz.
Honey, jam or jelly marmalade, 2 oz.

GENERAL INSTRUCTIONS

Eat slowly and chew thoroughly.

AVOID

Large meals and indigestible articles of food.
All fried foods, all foods made with fat and oils, cream, egg yolks.
Duck, goose, salmon, bacon, herring.
All pastries, Yorkshire and suet puddings.
Chocolate, cocoa, strong tea and coffee.
Condiments, spices and alcoholic beverages.

TAKE FREELY

Fresh uit and vegetables which should be sieved.
Hone jam and sugar.
Wate ınd fruit drinks. ean,
ale brea
marmalad

oc

DIET No. 22

Chron Interstitial Hepatitis without Jaundice (Cirrhosis of the Liver)

Iigh CHO. Moderate Fat. High Protein Diet.

App ximately: Prot. 130. Fat 70. CHO 410. Calories 2,780.

Diet No. 21 will meet these requir ments if 1¾ oz. grilled bacon or quivalent (see Exchange No. 1(*b*), p. 446) is taken at breakfast.

GENERAL INSTRUCTIONS

As for Diet 21.

Cholecystitis

Acute Cholecystitis

For dietetic instructions, see p. 304.

DIET No. 23

Chronic Cholecystitis (without obesity)

Approximately: Prot. 90. Fat 70. CHO 380. Calories 2,510.

EARLY MORNING Glass of water, hot or cold.

BREAKFAST 4 to 5 tablespoonfuls porridge (unless flatulence) or average helping cornflakes. 2 tablespoons syrup.
Milk from ration.
Small helping white fish, steamed or baked, with small helping tomato, or cold lean ham – all visible fat removed.
2 thin slices bread as crisp toast.
Butter (or vitaminized margarine) from ration.
Jelly marmalade, 1 dessertspoon.
Tea with milk from ration.

FORENOON Juice 1 orange.
Sugar from ration. } Glass of water, hot or cold.

DINNER Cup of Marmite.
Average helping lean meat – grilled or broiled, or rabbit, mutton, tripe, fish or fowl.
Sieved vegetable. (If flatulence, avoid cabbage, sprouts, stalk of cauliflower, peas, onion, turnip.)
Average helping potato, baked in skin and very floury.
Small helping milk pudding. fr
Stewed prunes or other fr[illegible]
Few boiled sweets.

3 P.M. Glass of water, ho[illegible]fuls porrid[illegible]

TEA Small helping ch[illegible] breakfast[illegible]it, white fish or l[illegible] meat, or 1 egg + 1 o[illegible]. skim milk cheese.
Average helping salad – tender lettuce leaves, t[illegible]omato, cress.
3 thin slices bread or crisp toast.
Honey or jam, 1 dessertspoon.
Tea with milk from ration.

SUPPER 1½ thin slices bread, or equivalent.
Honey or jam, 1 dessertspoon.
Banana or other fruit.
Tea with milk from ration.

BED-TIME Glass of hot water.

DAILY RATIONS Milk, whole, 1 pint.
Butter (or vitaminized margarine), 1¼ oz.
Sugar, 2 oz.

If 6 oz. skim milk replaces 6 oz. whole milk from ration, daily butter allowance can be increased to 1½ oz.

If flatulence is present, suitable vegetables are: tender, shredded lettuce, tomato, marrow, French beans, flower of cauliflower, asparagus, carrot, spinach.

GENERAL INSTRUCTIONS

Eat slowly and chew thoroughly.

AVOID OR TAKE SPARINGLY

All soups, especially cream soups.
Fried foods and foods baked in fat as Yorkshire pudding.
Foods made with much butter and eggs, suet puddings, pies and pastries.
Highly seasoned and twice-cooked foods.
Pork, duck, goose, salmon, herring, and fat of meat.

Brains, sweetbread, kidney, liver, and fish roe.
Toffee, caramels and marzipan.
Strong tea and coffee, cocoa, chocolate, alcohol.

ke plenty of fluid between meals.

DIET No. 23A. – More Expensive

Chronic Cholecystitis (without obesity)

Approximately: Prot. 90. Fat 70. CHO 380. Calories 2,510.

ARLY MORNING	Glass of water, hot or cold.
REAKFAST	½ grapefruit. Glucose from ration. Average helping porridge, grapenuts, cornflakes, Force or shredded wheat. Milk from ration. Small helping finnan haddock, or fillet of sole or other white fish. or Occasionally, lean, crisp, grilled bacon. 2 thin slices stale bread or crisp toast. 1 dessertspoon marmalade. Tea or coffee.
RENOON	Juice 1 orange. ,, ½ lemon. } Glucose from ration. Glass of hot water. or Cup of Marmite.
NCH	Average helping fillet of lean beef, mutton or fowl. Purée of celery, spinach or tomato. Average helping potato, baked in skin or mashed with a little skim milk. Banana baked, or fruit fool made with equal quantities custard powder sauce or thin arrowroot. No cream. 3 water biscuits. ½ oz. Cheddar cheese. Barley sugar or few glucose sweets.
P.M.	Fruit drink as above.
EA	2 slices brown bread and butter (or vitaminized margarine), or equivalent. Tomato pulp as sandwich.
INNER	Clear soup. Average helping chicken, rabbit, tripe or fish. Tomato, asparagus tips or tender string beans. Potato (baked in skin) or rice – average helping. Stewed fruit or fruit jelly – average helping. Curds with milk from ration. Coffee. Barley sugar or few boiled sweets.
ED-TIME	Fruit drink as above.
AILY RATIONS	Milk, whole, ½ pint. Milk, skim, ½ pint. Butter (or vitaminized margarine), 1½ oz. including cooking. Sugar and glucose, 3 oz.

If flatulence is present, suitable vegetables are: tender shredded lettuce, tomato, marrow, French beans, flower of cauliflower, asparagus, carrot and spinach.

GENERAL INSTRUCTIONS

Eat slowly and chew thoroughly.

AVOID OR TAKE SPARINGLY

All soups, especially cream soups.
Fried foods and foods baked in fat as Yorkshire pudding.
Foods made with much butter and eggs, suet puddings, pies and pastries.
Highly seasoned and twice-cooked foods.
Pork, duck, goose, salmon, herring and fat of meat.
Brains, sweetbread, kidney, liver, fish roe.
Toffee, caramels and marzipan.
Strong tea and coffee, cocoa, chocolate, alcohol.

Take plenty of fluid between meals.

DIET No. 24

Chronic Cholecystitis and Obesity

Approximately: Prot. 80. Fat 50. CHO 108. Calories 1,200.

EARLY MORNING	Glass of hot water.
BREAKFAST	Average helping white fish, steamed or baked, with small helping tomato, or cold lean ham (all visible fat removed). 1 thin slice bread or crisp toast. Butter (or vitaminized margarine) from ration. Tea with milk from ration. No sugar.
FORENOON	Juice 1 orange in glass hot water, or cup of Marmite.
DINNER	Small helping lean meat, all visible fat removed – grilled or broiled – or rabbit, tripe, fish or fowl. Sieved green vegetable. If flatulence, avoid cabbage, sprouts, stalk of cauliflower, peas, onion, turnip. Fruit jelly or stewed fruit. Curds with milk from ration.
3 P.M.	Glass of hot water.
TEA	Average helping chicken, rabbit, white fish or very lean meat, or 1 egg + 1 oz. skim milk cheese. Average helping salad, tender lettuce leaves, tomato, cress. 1 thin slice bread or crisp toast. Butter (or vitaminized margarine) from ration. Tea with milk from ration.
SUPPER	1 thin slice bread. Butter (or vitaminized margarine) from ration. 1 raw apple. Tea with milk from ration.

BED-TIME	Glass of hot water.
DAILY RATIONS	Milk, whole, 1 pint. Butter (or vitaminized margarine), ¾ oz. including cooking.

N.B. – For Dietetic Instructions, see Diet No. 23.

DIET No. 24A. – More Expensive

Chronic Cholecystitis and Obesity

Approximately: Prot. 80. Fat 50. CHO 108. Calories 1,200.

EARLY MORNING	Glass of hot water.
BREAKFAST	½ grapefruit. Saccharin if desired. Average helping grapenuts, cornflakes, Force or shredded wheat. Milk from ration. Average helping finnan haddock, or fillet of sole or other white fish. or Occasionally, crisp, grilled bacon or poached egg or scrambled egg if well tolerated. 1 thin slice stale bread or crisp toast, or 2 pieces Ryvita or Vita-Weat. Butter (or vitaminized margarine) from ration. Tea or coffee. No sugar.
FORENOON	Juice 1 orange in glass of hot water.
LUNCH	Small helping fillet of beef, mutton or fowl. Purée of spinach, tomato or other vegetable. Lemon snow or jelly. 2 water biscuits. ¾ oz. skim milk cheese.
3 P.M.	Glass of hot water.
TEA	3 water or breakfast biscuits or 3 tea rusks. Tomato. Butter (or vitaminized margarine) from ration. Tea with milk from ration.
DINNER	Small helping chicken, tripe or white fish. Tomato, asparagus tips or tender string beans. Stewed fruit or fruit jelly. Curds with milk from ration. Coffee.
BED-TIME	Hot fruit drink as above.
DAILY RATIONS	Milk, whole, 1 pint. Butter (or vitaminized margarine), ¾ oz. including cooking.

N.B. – For Dietetic Instructions, see Diet No. 23.

DIET No. 25

Acute Nephritis

Approximately: Prot. 5. Fat 25. CHO 190. Calories 1,000.

6 A.M. Juice 1 orange.
,, ½ lemon.
Lactose, 1 tablespoon.

8 A.M. Baked apple, average helping.
Caramel sauce (4 lumps sugar).
Double cream, 1 tablespoon.

10 A.M. Stewed pears, average helping.
Syrup sauce (colouring cochineal).

12.30 P.M. Lemon sago { ½ oz. sago.
Gill water.
Juice ½ lemon.
Syrup, 2 teaspoonfuls.
Decorate 1 tablespoon double cream.
Few boiled sweets.

2.30 P.M. Mashed banana, average helping.
Whipped cream, 1 tablespoon.

5 P.M. Tangerine orange slices.

7 P.M. Apple mould { Sieved apple, average serving.
4 tablespoons water.
1 teaspoon arrowroot.
1 teaspoon honey.
Decorate 1 tablespoon double cream.
Few boiled sweets.

9 P.M. Grape juice, ½ glass.
Lactose, 1 dessertspoonful.

If nausea or sickness still present, omit cream.

Note. – In place of ½ oz. cereal, may take instead any fruit except prunes and plums.

Half a pint of water may be taken during the day.

DIET No. 26

1. Acute Nephritis (i) Mild cases.
(ii) Severe cases, improving.
(iii) Focal nephritis.

2. Surgical Cases with Marked Nitrogen Retention – Initial Treatment.

3. Severe Chronic Nephritis with Marked Nitrogen Retention – Initial Treatment.

Approximately 1,800 Calories. Prot. 44 grams.

BREAKFAST Breakfast cereal as cornflakes, average serving.
Fruit, stewed or raw, average serving.

Wholemeal bread, 2 thin slices.
Marmalade.
Weak tea with milk and sugar from ration.

FORENOON Juice 1 orange.

DINNER Cheese macaroni or fish kedgeree, or other low protein dish. See Recipes on pp. 419 to 421.
Potato, average serving.
Green vegetable, average helping. Salad in season.
Milk pudding made from ½ oz. tapioca, cornflour or arrowroot. Milk from ration.
Fruit, fresh or stewed, average helping.

TEA 1 egg, or Exchange No. 1(*a*) (see p. 446).
Wholemeal bread, 2 thin slices.
Honey or jam, 1 dessertspoon.
Weak tea with milk and sugar from ration.

SUPPER Bread or equivalent, 1½ thin slices.
Jam, 1 dessertspoon.
Remainder of milk.

Instead of bread for supper, if patient can afford it, take preferably some fruit, *e.g.* apple, orange, banana.

DAILY RATIONS Milk, 10 oz.
Butter (or vitaminized margarine), 2 oz.
Sugar, 2 oz.

Seasoning in strict moderation.

Omit all salt if œdema is present; no pickles or condiments.

Total fluids, including milk and tea, not to exceed 1½ to 2 pints daily.

If desired, the caloric value can be increased by the addition of one or more 100-Calorie portions of carbohydrate or fat (see p. 443).

DIET No. 26A. – More Expensive

1. **Acute Nephritis** (i) Mild cases.
(ii) Severe cases, improving.
(iii) Focal Nephritis.

2. **Surgical Cases with Marked Nitrogen Retention – Initial Treatment.**

3. **Severe Chronic Nephritis with Marked Nitrogen Retention – Initial Treatment.**

Approximately 1,800 Calories. Prot. 44 grams.

BREAKFAST Average serving fruit, *e.g.* apple, stewed or raw.
1 egg, boiled, poached or scrambled.
Bread, 1 thin slice, or 2 sections Ryvita.
1 dessertspoon marmalade.
China tea or Kaffee Hag or Postum, with milk and sugar from ration.

FORENOON	Glass of barley water or ½ glass orange juice.
LUNCH	Fresh salad – lettuce, tomato, raw carrot, cress – average helping. French dressing – lemon juice, olive oil. St. Ivel cheese, 1 oz., or other 7½-gram protein dish (see Recipes, pp. 419 to 421). 2 sections Ryvita or Vita-Weat. Milk pudding from ½ oz. sago, cornflour or arrowroot, and milk and sugar from ration. Stewed apple or pear, average helping.
TEA	1 cup freshly infused China tea. 2 water biscuits. Butter from ration. Jam, jelly or honey.
DINNER	Vegetable milk soup. Spaghetti egg or other 7½-gram protein dish (see Recipes, pp. 419 to 421). Mashed potato, small helping. Butter. Curds with milk from ration. Stewed or fresh fruit, average serving. Cream from ration.
DAILY RATIONS	Milk, 10 oz. Butter (or vitaminized margarine), 1½ oz. 40 per cent cream ration, 2 oz. Sugar, 1½ oz.

Seasoning in strict moderation.

Omit all salt if œdema present. No pickles or condiments.

Total fluids, including milk and tea, not to exceed 1½ to 2 pints daily.

If desired, the caloric value can be increased by the addition of one or more 100-Calorie portions of carbohydrate or fat (see p. 443).

DIET No. 27

1. Acute Nephritis (improving).

2. Chronic Nephritis with Mild to Moderate Failure in Renal Concentrating Power.

Approximately 2,300 Calories. Prot. 60 grams.

BREAKFAST	Breakfast cereal as cornflakes – average serving. Fruit, stewed or fresh, average serving. Wholemeal bread, 2 thin slices. 1 egg, boiled, poached or scrambled. Marmalade. 1 cup weak tea with milk from ration.
FORENOON	Juice 1 orange.
DINNER	Cheese macaroni or fish kedgeree or other low protein dish (see pp. 419 to 421). Potato, average serving. Carrot or turnip or green vegetable, average serving. No peas or beans. Milk pudding from ½ oz. tapioca, cornflour or arrowroot.

Milk from ration.
Fruit, stewed or fresh, average serving.
Few boiled sweets.

Tea Small helping rabbit, chicken, meat or fish.
Wholemeal bread, 2 thin slices.
Honey or jam. 1 dessertspoonful.
1 cup weak tea with milk and sugar from ration.

Supper Wholemeal bread, 2 thin slices or equivalent.
Jam, 1 dessertspoonful.
Remainder of milk as milk, cocoa or Ovaltine.
Few boiled sweets.

Instead of bread for supper, if patient can afford it, take preferably some fruit, *e.g.* apple, orange, banana.

Daily Rations Milk, ¾ pint.
Butter (or vitaminized margarine), 2½ oz.
Sugar, 2½ oz.

If desired, the caloric value can be increased by the addition of one or more 100-Calorie portions of carbohydrate or fat (see p. 443).

DIET No. 27A. – More Expensive

1. Acute Nephritis (improving).

2. Chronic Nephritis with Mild to Moderate Failure in Renal Concentrating Power.

Approximately 2,300 Calories. Prot. 60 grams.

Breakfast Average serving fruit, stewed or raw.
Mushrooms baked in milk (when in season) or baked tomatoes on toast.
Wholemeal bread, 1 thin slice, or 2 sections Ryvita.
Jelly marmalade or honey, 1 dessertspoon.
China tea or Kaffee Hag or Postum with milk from ration.

Forenoon Glass of barley water or ½ glass orange juice.
or Tomato cocktail { 2 ripe tomatoes sieved. few drops lemon juice.

Lunch Fish soufflé or other 7½-gram protein dish (see Recipes, pp. 419 to 421).
Average helping vegetable or salad.
2 sections Ryvita or Vita-Weat.
Milk pudding made from ½ oz. sago, cornflour or arrowroot, with sugar and milk from ration. Add ½ egg.
Fruit, stewed, tinned or fresh, average serving.

Tea 1 cup freshly infused China tea.
3 rich tea biscuits. Butter from ration.

Dinner Cauliflower or tomato cream soup.
Small helping baked stuffed cod.
or Halibut with cheese sauce.
or Casserole of mutton with tomatoes and spaghetti.
or Small helping pork with apple sauce.
or Roast chicken with bread sauce.

or Roast mutton with red currant jelly and brown gravy.
Average helping vegetable.
Curds with milk from ration.
Stewed or fresh fruit.
Cream from ration.

Daily Rations	Milk, 1 pint. Butter (or vitaminized margarine), 2½ oz. 40 per cent cream, 2½ oz. Sugar, 2½ oz.

If desired, the caloric value can be increased by the addition of one or more 100-Calorie portions of carbohydrate or fat (see p. 443).

DIET No. 28

Chronic Nephritis with Œdema but without Failure in Renal Concentrating Power

Allowing 100 to 110 grams protein daily. Caloric value approximately 2,400.

Breakfast	Breakfast cereal, average serving. Fruit, fresh or stewed, average serving. 2 eggs or equivalent. Bread, 2 thin slices. 1 cup weak tea with sugar and milk from ration.
Dinner	Average helping (3 oz.) of meat, chicken or rabbit, or fish, 4 oz. Vegetables, average serving (no peas or beans). Potato, average serving. Milk pudding, small helping, made from ½ oz. cereal as cornflour, sago or arrowroot, and milk from ration. Fruit, stewed, tinned or fresh, average serving. 2 water biscuits. 1 oz. cheese.
Tea	Large helping fish or 1½ oz. cheese + 2 eggs. Vegetable or salad, large helping. Bread, 2 thin slices or equivalent. Sponge cake, 1 medium slice. Honey, jam or syrup, 1 dessertspoon. 1 cup tea with sugar and milk from ration.
Supper	Bread, 2 thin slices. or Bread, 1 thin slice, and average serving of fruit if can afford it. Milk from ration.
Daily Rations	Milk, ¾ pint. Butter (or vitaminized margarine), 2 oz. (unsalted). Sugar, 2½ oz.

N.B. – No salt to be used in cooking or at table.

Avoid

Bacon, corned beef, ham, meat extracts, sausages, salt fish, smoked haddock, kippers, salmon, sardines, shell-fish.

Fluid, including tea and milk, not to exceed 1½ pints daily.

DIET No. 28A. – More Expensive

Chronic Nephritis with Œdema but without Failure in Renal Concentrating Power

Allowing 100 to 110 grams protein daily. Caloric value approximately 2,400.

EARLY MORNING	Juice of 1 orange.
BREAKFAST	Cornflakes, average serving. Stewed or baked apple. 1 egg boiled, poached or scrambled. Bread, 2 thin slices. Marmalade, 1 dessertspoon. 1 cup weak tea or caffein-free coffee.
LUNCH	Average helping sole or other white fish, steamed. ,, ,, vegetable. ,, ,, potato, baked in skin. Fruit salad, orange, apple, banana, average helping. 2 water biscuits. Cheese, 1 oz.
TEA	1 cup freshly infused tea. 1 rich tea biscuit. 1 plain sponge cake.
DINNER	½ grapefruit. Cream of celery soup, 5 oz. Chicken and egg salad: Average helping chicken. 1 hard-boiled egg. Lettuce, beetroot and cucumber. Mayonnaise, 1 dessertspoon. or Large helping meat, rabbit or tripe and vegetables. Fruit, stewed or fresh. 20 per cent cream, 1 oz. Kaffee Hag.
DAILY RATIONS	Milk, whole, ¾ pint. Butter (or vitaminized margarine), 2 oz. (unsalted). Sugar, 2 oz.

N.B. – No salt to be used in cooking or at table.

AVOID

Bacon, corned beef, ham, meat extracts, sausages, salt fish, smoked haddock, kippers, salmon, sardines, shell-fish.

Fluid, including tea and milk, not to exceed 1½ pints daily.

DIET No. 29

High Protein Diet (Epstein) for Chronic Nephritis with Œdema but without Nitrogen Retention

Approximately: Prot. 145 to 150. Fat 70. CHO 316. Calories 2,500.

Breakfast
Fruit, fresh or stewed, average serving.
White fish (boiled or steamed) large serving, 5 oz.
1 egg.
Bread, 2 thin slices.
Butter or vitaminized margarine from ration.
Jam or marmalade, 1 dessertspoon.
1 cup of tea with milk and sugar from ration.

Dinner
Lean meat (all visible fat removed) or chicken or rabbit, 4 oz.
Green peas or beans, large serving.
Potato, 1 large, boiled.
Bread, 1 thin slice.
Milk pudding, small helping.
2 water biscuits.
1 oz. cheese.

Tea
White fish or tripe, large serving, 5 oz.
Bread, 2 thin slices or equivalent.
Jam or syrup, 1 dessertspoon.
1 slice sponge cake.
Fresh fruit.
1 cup tea with milk and sugar from ration.

Supper
Bread, 2 thin slices or equivalent.
Butter (or vitaminized margarine) from ration.
Cheese, 1 oz.

Daily Rations
Milk, ½ pint.
Butter (or vitaminized margarine), ¾ oz.
Sugar, 2½ oz.

Fluids not more than 1½ pints daily.
Food to be cooked and served without salt.
Butter to be salt-free.

DIET No. 29a. – More Expensive

High Protein Diet (Epstein) for Chronic Nephritis with Œdema but without Nitrogen Retention

Approximately: Prot. 145 to 150. Fat 70. CHO 316. Calories 2,500.

Early Morning
Orange cocktail { Juice 1 orange.
Pinch ground ginger.

Breakfast
Small bunch grapes or apple or pear.
2 eggs cooked any way desired.
Crisp toast, 1½ thin slices, or Ryvita or Vita-Weat, 3 sections.

Butter (or vitaminized margarine) from ration.
Honey or marmalade, 1 dessertspoon.
China tea freshly infused.

FORENOON Fruit as at breakfast.

LUNCH Grilled steak, 4½ oz.
Kidney beans and tomato, average helping.
1 baked potato.
Fruit whip (average helping sieved fruit, 1 egg white).
2 water biscuits.
1 oz. Dutch cheese.

TEA 1 thin slice brown bread or equivalent.
Butter (or vitaminized margarine) from ration.
Tomato, cress as sandwich.
1 cup China tea.

DINNER ½ grapefruit.
Sole or other white fish and tomato, large helping, 5 oz.
or Fillet of plaice, with oysters, average helping.
or Stuffed veal cutlets using 1 egg white, average helping.
or Roast fowl with veal stuffing, average helping.
or Cold chicken and lean ham, average helping.
Green peas, large helping.
Potato, average helping.
Pineapple meringue or other sweet.
2 water biscuits.
Parmesan cheese, 1 oz.
Kaffee Hag.

DAILY RATIONS Milk, ½ pint.
Butter (or vitaminized margarine), ½ oz.
Sugar, 2½ oz.

Fluids not more than 1½ pints daily.
Food to be cooked and served without salt.
Butter to be salt-free.

DIET No. 30

Gout – Diet during Attack

Low purine, low protein, low Calorie diet.

Approximately: Prot. 43. Fat 50. CHO 195. Calories 1,400.

EARLY MORNING Glass of hot water.

BREAKFAST Fruit, fresh or stewed, average serving.
Bread (white), 2 thin slices.
Butter (or vitaminized margarine) from ration.
Small teaspoon jelly marmalade.
Weak tea or Kaffee Hag. Milk from ration.

DINNER Egg, poached or scrambled, or small helping macaroni au gratin.
Vegetable (except asparagus, spinach).
Milk pudding, ½ oz. cereal, 5 oz. milk.
Fruit, average serving.
Bread (white), 1 thin slice.

Tea
1 egg or 1 oz. cheese.
White bread, 2 thin slices.
Butter (or vitaminized margarine) from ration.
A little jam if wished.
Sponge cake, small piece.
Weak tea or Kaffee Hag. Milk from ration.

Supper
White bread, 1 thin slice.
Milk from ration.

Daily Rations
Milk, 10 oz.
Butter (or vitaminized margarine), 1 oz.
Sugar, 1 oz.

Foods Permitted

Those practically purine-free, *e.g.* milk, butter (or vitaminized margarine), cheese, eggs, nuts, gelatine, sugar, white bread, cereals, fats, fruits (excepting strawberries), potatoes and other root vegetables, green vegetables (avoiding asparagus and spinach).

Foods Forbidden

Liver, kidney, sweetbread, excess of beef, mutton, bacon, veal, pork, fowl, game and fish, coffee, cocoa, alcohol, condiments and spices, meat soups and extractives, oatmeal, wholemeal bread, pulses, mushrooms.

Ample fluids should be taken between meals.

DIET No. 30A. – More Expensive

Gout – Diet during Attack

Low purine, low protein, low Calorie diet.

Approximately: Prot. 43. Fat 50. CHO 195. Calories 1,400.

Early Morning
Glass of hot water.

Breakfast
½ grapefruit or orange.
Savoury tomato – 1 egg baked in scooped-out tomato, or scrambled with tomato.
White bread, toasted, 2 thin slices.
Marmalade.
1 cup Instant Postum (1 level teaspoon).

11 A.M.
Hot or cold lemon drink.

Lunch
Salad in season, large helping.
Cauliflower au gratin, or cheese soufflé, or cheese macaroni.
Potato, small helping.
Fruit, fresh or stewed, average helping.

Tea
Bread, 1 thin slice, or 2 plain biscuits.
Butter (or vitaminized margarine).
Tomato, cress.
1 cup freshly infused China tea.

Dinner	Vegetable milk soup. Egg or cheese dish. Potato, small helping. Vegetable as salad, string beans, red cabbage or carrot. Fruit, stewed or fresh, or fruit jelly. Cream.
Daily Rations	Milk, 10 oz. Butter (or vitaminized margarine), ¾ oz. 20 per cent cream, 1 oz. Sugar, 2 oz.

Foods Permitted

Those practically purine-free, *e.g.* milk, butter (or vitaminized margarine), cheese, eggs, nuts, gelatine, sugar, white bread, cereals, fats, fruits (excepting strawberries), potatoes and other root vegetables, green vegetables (avoiding asparagus and spinach).

Foods Forbidden

Liver, kidney, sweetbreads, brains, excess of beef, mutton, veal, pork, bacon, fowl, game and fish, coffee, cocoa, alcohol, condiments and spices, meat soups and extractives, oatmeal, wholemeal bread, pulses, mushrooms.

Ample fluids should be taken between meals.

DIET No. 31

Chronic Gout

Restricted purine diet.

Approximately: Prot. 80. Fat 105. CHO 233. Calories 2,200.

Early Morning	Glass of hot water.
Breakfast	Fresh or stewed fruit, average serving. 1 egg cooked any way desired, but preferably not fried. White bread, 2 slices. Butter (or vitaminized margarine) from ration. Marmalade. Weak tea or Kaffee Hag. Milk from ration.
Dinner	Average helping lean meat or poultry or white fish. No glandular organs. Salad in season, large helping. or Other vegetable, avoiding those forbidden. Potato, average serving. Milk pudding, small serving. Fruit, average serving.
Tea	1 egg and ¾ oz. cheese or equivalent. White bread, 2 slices. Jam. Sponge cake, small piece. Weak tea or Kaffee Hag. Milk from ration.
Supper	White bread, 1½ slices. Milk from ration.

DAILY RATIONS	Milk, 1 pint. Butter (or vitaminized margarine), 2½ oz. Sugar, 1 oz.

Ample fluids to be taken between meals.

AVOID

Liver, kidney, sweetbread, brains, duck, pork, goose and excess of meat; highly seasoned foods and sauces, coffee, cocoa, alcohol, condiments and spices, meat soups and extractives.

Limit tea to 2 cups daily.

PERMITTED

Milk, butter (or vitaminized margarine), cheese, eggs, nuts, gelatine, fruits (excepting strawberries), sugar, potatoes and other root vegetables, green vegetables, *avoiding* asparagus and spinach. White bread, cereals and fat.

DIET No. 31A. – More Expensive

Chronic Gout

Restricted purine diet.

Approximately: Prot. 80. Fat 105. CHO 233. Calories 2,200.

EARLY MORNING	Glass of hot water.
BREAKFAST	Stewed fruit. Cornflakes or other breakfast cereal. 1 egg cooked any way desired, but preferably not fried. Tomatoes. White bread, 1 thin slice, may be toasted. Ryvita or Vita-Weat, 2 sections. Marmalade. 1 cup Instant Postum (1 level teaspoon).
11 A.M.	Hot or cold lemon drink.
LUNCH	Salad in season, large helping. Omelette. or Spaghetti and tomatoes, or macaroni and cheese, or cauliflower au gratin. Potato, average helping. Fruit, fresh or stewed, or fruit jelly. Biscuits and cheese.
TEA	Thin white bread and butter (or vitaminized margarine). Tomato, cress, sandwich. 1 cup freshly infused China tea.
DINNER	Vegetable milk soup. Small helping chicken, lean meat, rabbit, fish or tripe. Salad when possible, large helping, or Green or root vegetable, avoiding those forbidden. Fruit as at lunch. Cream. 2 water biscuits. ¾ oz. cheese.

Daily Rations	Milk, 1 pint. Butter (or vitaminized margarine), 1¼ oz. 20 per cent cream, 2 oz. Sugar, 1½ oz.

Ample fluids to be taken between meals.

Avoid

Liver, kidney, sweetbread, brains, duck, pork, goose and excess of meat, highly seasoned foods and sauces; coffee, cocoa, alcohol; condiments and spices; meat soups and extractives. Limit tea to 2 cups daily.

Permitted

Milk, butter (or vitaminized margarine), cheese, eggs, nuts, gelatine, fruits (excepting strawberries), sugar, potatoes and other root vegetables, green vegetables, *avoiding* asparagus and spinach. White bread, cereals and fats.

DIET No. 32

Obesity

Approximately: Prot. 70. Fat 36. CHO 100. Calories 1,000.

Early Morning	Glass of water.
Breakfast	Wholemeal bread, 2 thin slices. 1 egg. Tomato, average helping. Tea with milk from ration. Saccharin if desired.
11 a.m.	1 small apple or orange.
Dinner	Clear soup. Lean meat – all visible fat removed – average helping. Salad or green vegetable, large helping. Fruit, fresh or stewed, average helping. No sugar.
Afternoon	Glass of water.
Tea	White fish, small helping. Salad, average helping. Wholemeal bread, 2 thin slices. Tea with milk from ration.
Supper	3 water biscuits or equivalent. Tea or milk from ration.
Daily Rations	Whole milk, 2 oz. Skim milk, 8 oz. Butter (or vitaminized margarine), ¾ oz.

N.B. – Mineral oil (liquid paraffin) and lemon juice or vinegar may be used to make salad dressing.

No potatoes, sweets, jams, sugar, preserved or tinned fruits.

No fried foods.

No sweet wine, spirits, beer or sweet aerated waters.

If hungry, eat more vegetables.

Allowed ad lib.: Clear soup, Marmite, Bovril, Oxo, black coffee and tea, salad and green vegetables, raw fruit, saccharin.

See list of foods in descending order of caloric value (p. 444).

Above diet can be raised to 1,200 Calories by adding two 100-Calorie portions (see p. 443).

For instance, a medium-sized slice of bread (1⅓ oz.),
or 5 oz. potato, average helping,
or 6 oz. whole milk, 1 teacup.
Each yields approximately 100 Calories.

DIET No. 32A. – More Expensive

Obesity

Approximately: Prot. 70. Fat 36. CHO 100. Calories 1,000.

EARLY MORNING	Glass of water.
BREAKFAST	½ grapefruit. 1½ thin slices wholemeal bread, or 3 sections Ryvita. 1 egg or 1 oz. cold lean ham or tongue. Tea with milk from ration. No sugar.
11 A.M.	Glass of water.
LUNCH	Liver, kidney or sweetbread, small helping. or Very lean meat, chicken, tripe or fish (+ ¼ oz. butter), small helping. Potato, small helping. Raspberries, orange, greengages, average helping. or Stewed apple or pear, average helping. Curds with milk from ration.
TEA	Brown bread, moderate slice, or equivalent. Tomato, lettuce, cress, as sandwich. 1 cup tea with milk from ration. No sugar.
DINNER	Clear soup. Sole, or other white fish, small helping. or Very lean meat, chicken or rabbit (may be curried), small helping. Salad or other green vegetables, large helping. Potato, baked in skin, or mashed, small serving. Fresh fruit, as apricots, pear, apple or plums, average helping, or larger helping of same fruits stewed. Black coffee.
DAILY RATIONS	Milk (skim), 10 oz. Butter (or vitaminized margarine), ¾ oz.

N.B. – Mineral oil (liquid paraffin) and lemon juice or vinegar may be used to make salad dressing.

No potatoes, sweets, jams, sugar, preserved or tinned fruits.

No fried foods.

No sweet wine, spirits, beer or sweet aerated waters.

If hungry, eat more vegetables.

Allowed ad lib.: Clear soup, Marmite, Bovril, Oxo, black coffee and tea, salad and green vegetables, raw fruit, saccharin.

See list of foods in descending order of caloric value (p. 444).

N.B. – Above diet can be raised to 1,200 Calories by adding two 100 Calorie portions (see p. 443).

For instance, a medium-sized slice of bread (1½ oz.),
or 5 oz. potato, average helping,
or 6 oz. whole milk, 1 teacup.
Each yields approximately 100 Calories.

DIET No. 33

Severe Myocardial Failure

Low Calorie, low fluid, low salt diet.

Before Breakfast	Strained orange juice, 3½ oz. Glucose, ½ tablespoon. } Water to make 5 oz.
Breakfast, 8.30 a.m.	1 thin slice very crisp toast—buttered cold. Marmalade, jelly or honey. 1 small cup China tea. Milk and sugar (5 oz. fluid).
11.30 a.m.	Repeat fruit drink.
Dinner, 1.30 p.m.	Small helping of cream of chicken or rabbit or fish soufflé. 1 tablespoon cornflour sauce. Add a little Fresh tomato juice. Sieved spinach, 1 tablespoon.
Afternoon	5 oz. water or fruit drink as before.
Supper, 6 to 7 p.m.	Custard—5 oz. milk, 1 egg. or Benger's, Allenbury's, Farex or Ovaltine, 5 oz.

Total fluids as such in above dietary including milk and tea not to exceed 1½ pints.

No salt to be used in cooking or at table.

Patients must remain in bed whilst on this diet.

DIET No. 34

Mild Cases of Myocardial Failure

Calories approximately 1,500.

Early Morning	Orange juice, glucose.	
Breakfast	1 egg poached, boiled or scrambled. 1 thin slice crisp toast. Butter or vitaminized margarine. Honey, jelly or treacle. 1 small cup China tea or Postum or caffein-free coffee.	
10.30 a.m.	Orange and egg flip.	Beat egg yolk with 1 tablespoon milk and cream. Add glucose and strained orange juice. Lastly stir in stiffly beaten egg white.

DINNER	No soup. Small helping of tripe or chicken or rabbit. 1 tablespoon sieved cauliflower, spinach or carrot. Fairy toast with butter or Ryvita, Vita-Weat or very little creamed potato. Fruit whip, jelly or stewed or baked apple or sieved apricots with cream.
TEA.	1 cup weak freshly infused tea. Milk and sugar. 2 fingers of sponge cake or 2 plain biscuits.
SUPPER—NOT LATER THAN 7 P.M.	Small helping of white fish or lightly cooked egg or St. Ivel cheese. Thin bread and butter or vitaminized margarine. Light sweet as curds or custard, or If any indigestion substitute Benger's, Berina, arrowroot or Allenbury's diet.
DURING NIGHT	Orange juice and glucose.

If œdema present restrict salt and fluid (see Diet No. 33, p. 497).

DIET No. 35

High Carbohydrate, High Calorie Fluid Diet, for use in Fevers of Long Duration, *e.g.* Enteric Fever—Early Stages

Approximately: Prot. 76. Fat 137. CHO 360. Calories 3,000.

6 A.M.	Cup of tea freshly infused with Hot milk, 6 oz. Thin cream, 1 oz. Sugar, 2 lumps.
8 A.M.	Cereal such as Benger's, Allenbury's or Cream of Wheat ½ oz. Lactose 1 oz. Milk 6 oz. Thin cream 1½ oz. 1 egg.
10 A.M.	Egg and orange drink: Mix 1 egg yolk with cream, 1 tablespoon, and milk, 2 tablespoons. Add juice of 1 orange and ½ lemon. Add lactose 1 oz. and stir in beaten white of 1 egg.
12 NOON	Cream soup as spinach, celery, carrot, tomato purée. 1 teaspoon each butter (or vitaminized margarine) and cornflour. Add gradually 5 oz. milk, and cook well. Stir in 3 oz. sieved vegetable. Remove from fire and add 1 egg yolk. Add 2 oz. thin cream.
2 P.M.	Cocoa, 1 teaspoon. Milk, 6 oz. Thin cream, 1 oz. Lactose, 1 oz.
4 P.M.	Switched egg in 5 oz. milk. Thin cream, 1½ oz. Lactose, ½ oz.
6 P.M.	Creamed soup, as above, flavoured with Marmite.

8 P.M. Ovaltine, 2 heaped teaspoons.
Milk, 6 oz.
Lactose, ½ oz.

To sip during day and night: Juice of 2 oranges.
,, ,, 1 lemon.
Glucose, 2 oz.
Lactose, 2½ oz.
} Water to make 1½ pints.

DAILY RATIONS Milk, 2 pints.
20 per cent cream, ½ pint.

A total of at least 5 pints fluid should be taken daily.

To make good salt loss from sweating, salt should be added liberally to the cereal and soup feeds, and ⅓ teaspoonful should be added to the orange lemonade.

Should diarrhœa occur, give glucose in place of lactose.

DIET No. 36

Convalescence

Approximately: Prot. 83. Fat 125. CHO 300. Calories 2,650.

MORNING Juice of 1 orange, with sugar to taste.

BREAKFAST Breakfast cereal, small serving, with cream, and 1 or 2 tablespoons of Bemax.
Egg – boiled, poached or scrambled.
Toast, 2 thin slices.
Weak tea, with milk and sugar.

MID-FORENOON Glass of milk.
¼ to ½ teaspoonful Marmite.
2 cream crackers.

DINNER Steamed white fish.
or Minced chicken or rabbit.
or Sweetbreads or tripe.
Potatoes – boiled or mashed with cream.
Vegetable purée, or cauliflower, or sieved creamed carrots.
Milk pudding, custard or milk jelly.
Stewed fruit.

TEA Egg – boiled, poached or scrambled, or white fish.
Bread, 2 slices.
Sponge cake, 1 slice.
Weak tea, with milk and sugar to taste.

SUPPER Biscuits.
Cocoa, or milk food (Horlick's, Ovaltine, Farex, etc.).

DAILY RATIONS Milk, 30 oz. (1½ pints).
Butter (or vitaminized margarine), 2 oz.
Cream, 5 oz.
Sugar, 2 oz.
Jam, 2 oz.

DIET No. 37

Chronic Pulmonary Tuberculosis

Approximately: Prot. 113. Fat 170. CHO 330. Calories 3,300.

MORNING	Orange juice, with sugar to taste.
BREAKFAST	Porridge or breakfast cereal. Bacon and egg, or fried fish, or omelette. Bread or toast, 2 slices. Tea with milk and sugar to taste.
FORENOON	Glass of milk.
DINNER	Cream of potato or other thick soup. Average serving of meat or fish. Potatoes, fried, sauté, or mashed with cream. Vegetables with butter (or vitaminized margarine), or salad with mayonnaise. Steamed pudding, or custard, or milk jelly. Stewed fruit, if desired.
TEA	Bread, 2 slices. Cake, 1 slice. Tea with milk and sugar to taste.
SUPPER	Egg or cheese soufflé, or fish. Bread, 2 slices. Biscuits or cake. Glass of milk.
BED-TIME	Milk drink, *e.g.* Ovaltine, Horlick's. Biscuits.
DAILY RATIONS	Milk, 2 pints. Butter (or vitaminized margarine), 2 oz. at least. Cream, 2 oz. at least. Sugar, 2 oz. Jam, 2 oz.

DIET No. 38

For Normal Child of Five Years

Wt. = 40 lb. Allow 40 Calories per pound of body weight.

Approximately: Prot. 70. Fat 70. CHO 200. Calories approx. 1,700.

BREAKFAST	Small helping of stewed figs or prunes or raw apple or orange. Porridge, grape nuts, Farex, cornflakes or shredded wheat. 1½ slices crisply toasted brown bread. Butter when cold. 1 egg – boiled, poached or scrambled. Honey or marmalade. Certified milk (cocoa or tea may be used to flavour).

DINNER

Milk vegetable soup (carrot, tomato).
Small serving meat, mutton, chicken, fish, rabbit, tripe lightly cooked, liver or herring (two latter being especially valuable).
Always green vegetables, particularly spinach and salad including puréed tomato and a little grated raw carrot.
Small potato, baked in skin.
Always fruit – stewed or raw. Avoid unripe and over-ripe fruit.
Curds or milk to drink – occasionally milk pudding or custard, using breakfast egg.
Few boiled or glucose sweets.

SUPPER

1½ slices wholemeal bread, crisp toast, Ryvita, Vita-Weat or plain biscuits.
½ oz. cream cheese, or fish, or egg dish.
Honey or treacle.
Milk to drink.

DAILY RATIONS

1½ pints certified milk.
(cheaper milk may be used for cooking.)
1 oz. butter (or vitaminized margarine).
1 oz. sugar or glucose.
3 glasses water.

AVOID

New bread, scones, cakes, pastry and highly seasoned foods.
Sweets at night and before meals.
Tea and coffee (except for flavouring milk).

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